University of Alberta

Direct and Indirect Determinants of Low Birth Weight in a Large Canadian Urban Health Region

by

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Abstract

Low birth weight (LBW) is the single most important determinant of perinatal mortality, and infant and childhood morbidity. With increasing rates of LBW and preterm births (PTB), prevention is a major public health priority. The primary purpose of this study was to determine the direct and indirect effects of maternal age, genetic and constitutional, socio-economic, obstetrical, medical, nutritional, prenatal, and lifestyle risk factors on birth weight and gestational age in a large urban health region in Alberta, Canada. In addition, this study used mapping techniques to describe the geographical distribution of risk factors and birth outcomes.

A retrospective population-based cohort study was conducted using computerized administrative databases. The study population included women who delivered a singleton live birth with no major fetal anomalies between 1996 and 1998 (N=26,265). Multiple logistic regression was used to estimate the effects of the risk factors on PTB and LBW.

The study results demonstrate that the identified risk factors had a direct and/or indirect effect on birth weight and gestational duration. Factors having a direct effect on PTB and LBW included pre-existing maternal diseases, poor obstetrical history, medical problems during pregnancy, pregnancy complications, and inadequate prenatal care. In the LBW model, smoking during pregnancy, poor gestational weight gain, and anemia also had direct effects on birth weight. Among socially disadvantaged women, low pre-pregnancy weight and street drug use adversely influenced birth weight. Women who smoked and consumed alcohol during pregnancy were at an increased risk for delivering a preterm infant. Socio-economic status and maternal age had an indirect effect on both birth outcomes. Mapping of the geographical distribution of risk factors and birth outcomes revealed that areas that had high PTB and/or LBW rates did not always have corresponding high risk factor prevalences.

Identification of the distribution and determinants of PTB and LBW should assist health providers to develop more effective primary and secondary prevention interventions. The results of the study suggest that innovative approaches are required to increase utilization of prenatal care services and that prevention strategies need to be directed towards lifestyle behaviors and early detection and treatment of medical and pregnancy complications.

DEDICATION

In Loving Memory of My Parents Clarence and Sarah Knox

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Chapter 1 Introduction

Statement of Problem

Birth weight is an important infant health indicator because it predicts infant survival and future health status (Alberta Health 1996; Kramer, 1987). Low birth weight (birth weight less than 2500 grams) is a major determinant of neonatal mortality, as well as infant and childhood morbidity (Hack, Lein, & Taylor, 1995; Paneth, 1995). Low birth weight (LBW) may occur as a result of being born too soon (preterm birth (PTB)), born too small (small for gestational age (SGA)), or a combination of both processes. Preterm infants comprise approximately 65-70% of LBW infants (Joseph, 1998).

LBW infants, specifically preterm LBW infants, are of concern to health professionals and policy makers because they contribute to 75-85% of all neonatal deaths and 50% of long-term neurological impairment in children (Alexander, Dodds, & Armason, 1998; Committee to Study the Prevention of Low Birth Weight, 1985; Joseph, 1998; McCormick, 1985; Moutquin & Papiernik, 1990). These infants are at greater risk for neuro-developmental handicaps, chronic respiratory problems, visual, and hearing deficits (Hogan & Park, 2000; Kramer, 1997; Nault, 1997; Zahr, 1999); are more likely to require special education services and social services (McCormick, 1985; Petrou, Sach, & Davidson, 2001); and, are more likely to consume health services into childhood (Lewitt, Schurman, Baker, Corman, & Shioni, 1995; Moutquin & LaLonde, 1998; Svensen & Schopflocher, 1997).

The economic consequences of LBW are also significant. The average cost of caring for LBW babies in Canadian hospitals (based on NICU per diem hospital costs) in 1995 has been conservatively estimated at $83,142 per infant (Moutquin & LaLonde, 1998). It has been reported that "every LBW preterm infant born in Canada imposes an economic burden to the health care system of about $40,000 up to one year of age and shares an actuarial cost of $678,000 for those with a permanent handicap of neonatal origin" (Moutquin & LaLonde, 1998, p. 6). This results in a health care expenditure of approximately 13.3 billion dollars per year. Researchers have suggested that if successful interventions could reduce the crude rate of prematurity by 20%, then there would be a corresponding annual savings of close to 2 billion dollars (Moutquin & LaLonde, 1998).

LBW rates in Canada (5.7%) and the United States (7.6%) are higher when compared with countries such as Finland (4%), Sweden (4.4%) and Switzerland (5.5%) (Best Resource Centre, 1998; United States Census Bureau, 2000). Although the LBW rate in Alberta (6.2%) is not the highest in Canada (e.g., Northwest Territories (6.7%); Nunavit (7.5%)), it is higher than some other provinces including British Columbia (5.1%), Manitoba (5.8%), Nova Scotia (5.6%), New Brunswick (5.4%), and Saskatchewan (5.8%) (Statistics Canada, 2000). The lower rates both internationally and provincially suggest that the LBW rate in Alberta (6.2%) may be reduced through prevention programs that target modifiable risk factors.

LBW has been identified as a major public health priority in Alberta (Alberta Health and Wellness, 1999; Alberta Health, 1996). Between 1994 and 1996, 4 of the 17 Regional Health Authorities (David Thompson, Keeweetinok Lakes, Calgary, and Capital Health) had a LBW rate above both the provincial rate of 6.2% (Tough, Svenson, & Schopflocher, 1999) and the national rate of 5.7% (Statistics Canada, 2000). Furthermore, there were wide geographical differences with respect to LBW within specific Regional Health Authorities (RHA). For example, in the Capital Health Authority (CHA) between 1996 and 1998, the LBW rate ranged from a low of 4.8% in South Central Public Health Service Area (PHSA) to a high of 8.8 % in Central PHSA (Capital Health Technical Report, 1998). The PHSA LBW rates have not decreased over time. Between 1997 and 1999, both South Central and Central PHSA rates increased to 5.4% and 8.9% respectively (Capital Health Technical Report, 2000).

Changes in the LBW rate often parallel increases in the incidence of PTBs (Joseph, 1998; Kramer, 1998). In Alberta, between 1994 and 1996, PTBs comprised about 55% of the 1994 and 1996 total LBW births (Tough et al., 1999), whereas, in 1998, PTBs comprised 67.8% of LBW infants. In Alberta, the PTB rate increased from 6.7% in 1993-1994 to 7.4% in 1996-1998 (Health Surveillance, 1999).

As a result of both the increasing LBW and PTB rates and the associated social, health, economic, and personal costs, the Deputy Minister of Health in Alberta established a provincial target of lowering the LBW rate from 6.2% to 5.5% by the year 2002 (Alberta Health, 1996; Tough et al., 1999). This target has not yet been achieved. However, RHAs are still committed to achieving this target, and as a result, it continues to be incorporated into the business plans of RHAs including Capital Health (Capital Health Technical Report, 2002). In order to meet this target, it is necessary to identify the modifiable risk factors for PTB and LBW. This information could then be used by practitioners and policy makers within CHA to develop and implement programs designed to reduce the LBW rates within the PHSAs of CHA.

To date, analysis of risk factors for both PTB and LBW in CHA and other RHAs has been completed for only a limited number of risk factors such as smoking, alcohol consumption, street drug use, and non-attendance at prenatal classes (Tough et al., 1999). In addition, there is limited information on risk factor prevalences (e.g., smoking, maternal age) within the PHSAs of CHA. Consequently, it is not known whether the variations in the LBW rates within CHA are related to geographic variations in the prevalence of the risk factors that affect either gestational duration and/or fetal growth.

A critical piece of the health surveillance within Alberta to be completed is to determine the predictors of PTB and LBW. This initial work has begun. Newburn-Cook, White, Svenson, Demianczuk, Bott, and Edwards (2002), acknowledging that PTB and fetal growth restriction are different pathways to LBW, used an electronic perinatal database to examine the relationship between 33 maternal risk factors (e.g., maternal age, genetic and constitutional factors, obstetrical history, lifestyle, pre-existing medical problems, pregnancy-related complications, medical problems during pregnancy, and nutritional problems) and the increased risks for preterm delivery and small for gestational age births (SGA) in a population-based study of 76,444 Alberta women in Central and Northern Alberta. However, this study did not examine the influences of either prenatal care factors or socio-economic factors, and did not provide specific analysis of risk factors by RHA or by the PHSAs of CHA. Furthermore, this study did not detail specifically the indirect and direct effects of the study factors on gestational and/or birth weight.

Purpose of the Study

The primary purpose of this study was to examine the association of a large number of previously identified risk factors on LBW (defined as an infant who weighs less than 2500 grams) and PTB (defined as an infant who is born prior to 37weeks gestation).

The objectives of the study were: a) to examine the simultaneous impact of previously identified risk factors (i.e., maternal age, genetic and constitutional factors, obstetrical history, lifestyle, pre-existing medical problems, pregnancy-related complications, medical problems during pregnancy, nutritional problems, inadequate prenatal care) on birth weight (LBW) and gestational age (PTB); b) to identify the direct and indirect effects of these risk factors on birth weight and gestational age; and, c) to illustrate and report the differences in the prevalences of risk factors within each of the PHSAs in CHA.

Significance of the Study for Nursing

This study is guided by an epidemiological framework, which seeks to determine the distribution and determinants of health outcomes from a population perspective. Epidemiology as a discipline has as its goals to understand and prevent disease, to promote health at the individual, aggregate, and population level, and to contribute to the development of health services and health policy (Mackenbach, 1995; Pierce, 1996; Valanis, 1999). These goals are congruent with the goals and actions of nursing.

Understanding the factors that contribute to health outcomes has been advanced with the recent determinants of health discourse (Evans, Barer, & Marmor, 1994; Federal, Provincial, and Territorial Advisory Committee on Population Health, 1994; Hamilton & Bhatti, 1996; Health Canada, 1996; Link and Phelan, 1995; Wilkinson, 1996). This discourse has expanded our understanding of what determines health and illness by identifying a broad range of determinants that influence health: social, economic, and physical environments; personal health practices and coping skills; health services; genetic and biological factors; gender and culture; and healthy child development.

Healthy child development has been singled out as a major health determinant given its impact on subsequent health status (Health Canada, 1996). It is also recognized that healthy child development is influenced by most other determinants. Moreover, there is increasing evidence to suggest that what happens to the unborn child in utero affects fetal development, neonatal and infant health, as well as adult health status (Federal, Provincial, and Territorial Advisory Committee on Population Health, 1994; Godfrey & Barker, 2000, Keen, 1993). Recent research suggests that several of the major diseases of later life, including coronary heart disease, and type 2 diabetes originate in impaired intrauterine growth and development (Godfrey & Barker, 2000; Godfrey & Barker, 2001; Leon, 2001). To reduce the morbidity and mortality associated with these adverse birth outcomes, there has been an increasing emphasis on identifying both proximal and distal factors that influence birth outcomes (Misra, O'Campo, & Strobino, 2001; Myslobodsky, 2001).

This study will provide information on the distribution of risk factors and adverse birth outcomes (LBW and PTB) within one health region. By identifying the direct and indirect effects of biological and genetic factors, personal health practices, socio-economic factors, and health service factors on PTB and LBW, this study will increase our understanding of *how* these factors operate to influence birth outcomes.

Epidemiological studies such as this one are significant for nursing for several reasons. Nurses work with families in many settings and at different time periods (preconceptually, prenatally, and postnatally). Consequently, nurses are in a key position to work with families and collaboratively with other health professionals to influence birth outcomes. Understanding the factors associated with preterm and low birth weight infants will enable nurses to develop effective primary prevention interventions that address modifiable risk factors that can influence birth outcomes. As well, secondary prevention strategies can be employed to ensure early detection and prompt treatment of pregnancy complications and existing medical conditions. Nurses are in a key position to address these factors, given their skills and knowledge in the health promotion strategies of creating supportive environments, development of personal skills, community development, and advocating for healthy public policy (Reutter, 2001). This study will also increase our understanding of the distribution of risk factor prevalences and birth outcomes in a large Regional Health Authority. This information can be used in planning effective allocation of resources, targeting services in at-risk areas or providing services across the entire health region. Finally, the study findings will provide baseline data that can be used in the identification of trends to predict future health care needs and to evaluate existing programs and policies that aim to enhance birth outcomes.

Epidemiological methods are becoming more important to health professionals, including nurses, as health system priorities shift from a focus on treatment to illness prevention (Brunt & Shields, 2000). Nevertheless, the role of nurse scholars in the identification of determinants of health is relatively new. Butterfield (2002) states that "with few exceptions, nursing has not been active in efforts to understand the etiology of disease" (p. 33). She contends, that nurses have a key role in advancing upstream thinking through research addressing etiology of disease, and that nurses can and "should be directing research addressing the etiology of disorders that impact our clients"(p. 45). Nurse scholars suggest that in order to effect changes in LBW trends, nurses need to understand the etiology of LBW so they can work with others to develop and implement strategies to reduce the incidence of LBW (Arnold & Grad, 1996).

Chapter 2 Review of the Literature

Assessment of the risk factors for LBW is difficult because: a) birth weight is dependent on two processes: gestational duration and the rate of intrauterine growth; and b) many etiologic risk factors interact in complex ways, and can affect one, or both, of these processes (Kramer, 1987). Thus, synthesis and analysis of the LBW research-based literature is complicated by the diversity in how investigators have defined LBW (e.g., birth weight less than 2500 grams, term SGA infants, preterm SGA infants), whether the measurement of risk factors has been complete and reliable, and the researchers' ability to control for confounders (Kramer, 1987).

Kramer's (1987) systematic review and meta-analysis of the English and French medical literature published between 1970-1984 provided a structure and a starting point in identifying which risk factors were associated or not associated with LBW and PTB. Kramer's assessment was restricted to singleton pregnancies of women with no chronic illnesses because PTB may be the result of early medical intervention arising from maternal and/or fetal problems during pregnancy. Multiple pregnancies were not included because these pregnancies are subject to growth restriction and preterm delivery. Medical complications during pregnancy were also excluded because complications were considered intermediate in the causal chain. That is, a risk factor (e.g., genital tract infection) may lead to a complication during pregnancy (e.g., premature rupture of membranes), which in turn may result in premature labor and delivery. Thus, control for premature rupture of membranes would diminish and might even eliminate the direct or indirect effect of infection on the birth outcomes. A total of 921 relevant publications were identified, of which 895 were located and reviewed. Kramer used a set of *a priori* methodological standards (i.e., control for confounders, description of population sample, description of participation and follow-up rate, time sequence between factor and outcome, and study design) to determine the independent effects of 43 risk factors on birth weight, gestational age, prematurity, and intrauterine growth restriction (IUGR). These factors were categorized into the following groupings: genetic and constitutional factors, demographic and psychosocial factors, obstetric factors, nutritional factors, maternal morbidity during pregnancy, toxic exposures, and antenatal care.

In this literature review, Kramer's meta-analysis was used to identify risk factors that had a potential impact on birth weight and gestational age. The study literature review was expanded to include more recent research studies that examined the previously identified risk factors, as well as additional variables, and that also addressed the methodological issues identified by Kramer (i.e., control for confounders).

This chapter is organized according to the different risk factors including a critical review of the evidence regarding their association with LBW and PTB. Based on the literature review, the chapter ends with the presentation of a hypothetical model of risk factors for LBW, which guided the analyses.

Risk Factors Associated with LBW and PTB

Maternal Age

Research studies that have examined the effect of maternal age on PTB and LBW have been inconclusive and often contradictory. Although some researchers have found an association between older maternal age (35 years of age or older) and PTB (Aldous & Edmonson, 1993; Ancel et al., 1999; Berkowitz et al., 1998; Berkowitz, Blackmore-Prince, Lapiniski, & Savitz, 1998; Chumnijarakij, Chitinand, Quamkul, Viputsiri, Limpongsanurak, & Thaineau, 1992; Meis et al., 1995; Newburn-Cook et al., 2002), other studies have found no association (Abrams, Newman, & Parker, 1989; Kramer, McLean, Eason, & Usher, 1992). Similarly, some researchers have found an association between young maternal age (less than 20 years of age) and PTB (Ancel, Saurel-Cubiizolles, Di Renzo, Papiernik, Breart, 1999; Berkowitz, Blackmore-Prince, Lapiniski, & Savitz, 1998; Fraser, Brockert, & Ward, 1995; Gortzak-Uzan, Hallak, Press, Katz, Shoham-Vardi, 2000; Michelliutte et al., 1992; Wessel, Cnattingius, Bergstrom, Dupret, & Reitmaier, 1996) and others have not (Kramer et al., 1992; Mercer, Goldenberg, Das, Moawad, Iams, & Meis, 1996). Investigators who have controlled for several age-dependent confounders (i.e., smoking, pre-pregnancy weight or BMI, weight gain) have found a significant association between PTB and maternal age 17 years of age or less (Abrams et al., 1989; Wen et al., 1990).

With respect to LBW, several investigators have reported an increased risk of LBW for younger women (less than 20 years of age) (Fraser et al., 1995; Gortzak-Uzan et al., 2000; Michelliutte et al., 1992; Wessel et al., 1996), and older women (35 years of age or older) (Chumnijarakij et al., 1992). However, these investigators did not control for socio-demographic factors (Chumnijarakij et al., 1992; Fraser et al., 1995; Wessel et al., 1996), lifestyle factors (Gortzak-Uzan et al., 1992), prenatal care use (Chumnijarakij et al., 1992; Wessel et al., 1996), or pregnancy complications. The odds ratios for younger women were in the range of 1.49 to 3.7, whereas the odds ratio for older women was 1.75.

Berkowitz and Papiernik (1993) suggested that the inconsistent findings regarding maternal age may reflect a small sample size, the number of risk factors and confounders examined jointly, and how the birth outcome is defined (i.e., PTB has been defined as 37 weeks completed gestation, medically indicated PTB (a PTB that occurs as a result of pregnancy complications or medical disorders) or spontaneous PTB (a PTB that occurs as a result of spontaneous onset of preterm labor or preterm premature rupture of membranes)) (Ancel et al., 1999; Berkowitz et al., 1998). Berkowitz et al.'s (1998) study (N=31,107) examined various risk factors (i.e., sociodemographic, lifestyle, medical, maternal and infant characteristics, and reproductive factors) and found different maternal age effects depending on how the birth outcome was defined. Younger women (20 years of age or younger) had a 50% greater risk for spontaneous preterm labor but were not at increased risk for preterm premature rupture of membranes. Older women (35 years of age or older), on the other hand, had a 50% greater risk for preterm premature rupture of membranes but were not at increased risk for spontaneous preterm labor. Meis et al. (1995), in their study of 26,205 women in Wales, showed that younger women did not have an increased risk (OR=1.17; CI: 0.71-1.91) for medically indicated preterm deliveries. However, for spontaneous preterm deliveries, younger women less than 18 years old and younger women 18 to 19 years old had a higher risk, odds ratios were 2.0 (CI: 1.43-2.81) and 1.54 (CI: 1.21-1.97) respectively. In contrast, older women had an 83% (OR=1.83; CI: 1.31-2.55) higher risk of medically indicated PTB and did not have an increased risk for spontaneous preterm delivery. Lang et al. (1996), in a study of 23 risk factors (N=9,490), did not report an increased risk for preterm labor for younger women (i.e., women 15 years or less and women 16 to 19 years of age) or older women (i.e., women 35 years or older); however, women 20 to 24 years of age were at increased risk (OR=1.9; CI: 1.5-2.3). The results of these studies suggest that there are different pathways associated with PTB and that etiological studies need to quantify the impact of different risk factors by PTB subtype (Lang et al., 1996). Furthermore, the studies suggest that risks for adverse outcomes may also differ according to how maternal age is operationally defined and the type and inclusiveness of the risk factors examined.

It is not clear if chronological age is truly a risk factor for PTB and LBW or whether the increased risk is reflective of the characteristics related to the extremes of age (e.g., increased medical problems, increased risk for pregnancy complications). For example, young maternal age (less than 20 years of age) may indirectly affect gestational age and birth weight because it has been shown that younger women are more likely to have inadequate gestational weight gain, initiate prenatal care late in their pregnancy, and engage in other lifestyle behaviors such as cigarette smoking, alcohol use, and recreational drug use that have been associated with poor pregnancy outcomes (Ekwo & Moawad, 2000; Ng & Wilkins, 1994; Strobino, Ensminger, Kim, & Nanda, 1995).

Researchers have suggested that the increased risk for PTB and LBW for older mothers results from a combination of factors. Older women are more likely to use fertility enhancing techniques resulting in multiple gestations (Joseph, Kramer, Marcoux, Ohlsson, Wen, Allen, & Platt, 1998), and are more likely to have pre-existing medical problems (e.g., hypertension). Older women also have an increased prevalence of pregnancy complications such as gestational hypertension (Ziadeh & Yahaya, 2001), trimester-specific bleeding (Jolly, Seibre, Robinson, & Regan, 2000; Zeitlin, Ancel, Saurel-Cubizolles, & Papiernik, 2001; Ziadeh & Yahaya, 2001), placenta previa, and placental abruption (Jolly et al., 2000; Prysak, Lorenz, & Kisly, 1995). This suggests that older maternal age (35 years of age or older) may increase the impact of other risk factors (Petridou, Salvanos, Skalkidou, Dessupris, Moustaki, & Trichopoulos, 2001), or that age is associated with the occurrence of these risk factors (Jolly et al., 2000; Prysak et al., 1995). Investigators who have controlled for age-dependent confounders have not provided consistent findings regarding the impact of maternal age on poor birth outcomes. These differential findings may be a result of the risk factors and confounders that were examined, definition of birth outcomes, and the operationalization of younger and older maternal age. None of the maternal age studies reviewed examined the indirect effects of age on gestational age and/or birth weight.

The findings regarding the effect of maternal age on PTB and LBW remain equivocal. Whether investigators reported a maternal age effect was dependent on the conceptualization of younger and older (advanced) maternal age, the age of the reference group chosen for comparison, whether age-dependent confounders were controlled, and how the birth outcome PTB and LBW were operationalized. PTB has been defined in studies as a birth less than 37 weeks gestation or by PTB subtype (i.e., preterm labor premature rupture of membranes, medically indicated PTB). Similarly, LBW has been operationalized as an infant born with a birth weight less than 2500 grams or an infant who was small for gestational age (i.e., a birth weight less than the tenth percentile according to gestational age).

More research is required to quantify the effects of maternal age on different birth outcomes. Researchers will need to consider a number of factors in the design of these studies. These include: a) what constitutes the definitions of older and younger maternal age; b) what is the optimal age for reproduction for age-specific comparisons; c) definition of birth outcomes and specific maternal age differences by PTB subtype and SGA births; d) control of age-dependent confounders (e.g., SES, parity, smoking, alcohol consumption, antenatal care); and e) inclusion of women with pre-existing diseases and pregnancy complications.

Socio-economic Status

The well-known pattern of an inverse relationship between socio-economic status (SES) and health has generally been found regardless of what measures of SES are used, what outcomes are studied, and when and where the analyses are done (Kaplan, 1996). Several explanations have been proposed to explain the mechanisms through which SES influences health. Theoretical explanations, as originally identified in the UK Black Report that specifically apply to women of childbearing age are the behavioral/cultural explanation and the materialist /structuralist explanation (Department of Health and Social Security, 1980).

One explanation of why SES differentials in health exist is the behavioral/cultural explanation. This approach suggests that those individuals in the lower social hierarchy have poorer health because they engage in more health-inhibiting behaviors, such as smoking, substance abuse, inadequate nutrition, and lower use of preventive health services. This explanation does not however acknowledge the influence of the social context in which individuals live and work, but rather implies that these behaviors are the result of individual free-choice decisions (Reutter, 2000).

In contrast to the behavioral/cultural explanation, the materialist/structuralist explanation of social inequities in health does recognize the influence of the social context on health outcomes and is concerned with the effects that social structures, rather than individual behaviors, have on health outcomes (Reutter, 2000; Williams, 1990). This explanation emphasizes that poor health results from decreased access to material prerequisites and resources that facilitate health (Rutter & Quine, 1990). This conceptualization acknowledges that poor health may result from unhealthy behaviors; however, it is further recognized that these behaviors are influenced by the social context in which people live and work (Reutter, 2000).

The medical and public health literature includes a number of studies that attempt to elucidate the relationship between SES and an increased risk of LBW and PTB throughout various parts of the world (Kogan, 1995; Parker, Schoendorf, & Keily, 1994). Consistently, the research demonstrates an increased prevalence of LBW among low-income women (e.g., lowest income quintile, below the poverty line) (Gazmarian, Adams, & Pamuk, 1996; Gudmundscon, Bjorgvinsdottir, Molin, Gunnarson, & Marsal, 1997; Kramer, Sequin, Lydon, & Goulet, 2000; Sanjose & Roma, 1991) and poorly educated women (e.g., less than high school, less than grade 9 education) (Chance & Walker, 1998; Parker et al., 1994). For example, in Canada, LBW rates are approximately twice as high for families in the lowest income quintile compared to families in the highest income quintile (Chance &Walker). PTB rates also vary according to the wealth of the neighbourhood. The rate of PTB is 7.4% in the poorest Canadian neighbourhoods and 5.7% in the richest (Wilkins, Sherman, & Best, 1991). In Canada, the number of individuals living in poverty has risen in the past decade from a rate of 14% in 1989 to a rate of 16.2% in 1999 (National Council of Welfare, 2002). Furthermore, a substantial percentage of childbearing women continue to live in poverty (National Council of Welfare, 2002), which increases their risk for adverse birth outcomes.

Understanding the pathways and mechanisms by which SES affects LBW and PTB is a challenge in public health research. SES is closely related to other demographic, behavioral, environmental and medical factors that may influence pregnancy outcome. Thus, SES is often considered a proxy for these factors (Berkowitz & Papiernik, 1993). While it has not been demonstrated definitively how SES contributes to LBW, several investigators have concluded that poverty exerts its influence on LBW through deprivation of the prerequisites of health that affect the woman's health prior to and during her pregnancy (Berkowitz & Papiernik, 1993; Kogan, 1995; Kramer, 1987; Rutter & Quine, 1990). Kramer (1987) reviewed 113 studies that examined the effect of socio-economic status on gestational duration and birth weight. The results suggest that SES does not have an independent effect on PTB, SGA or LBW; rather, low socio-economic status may exert influence through nutritional, lifestyle, anthropometric, and infectious factors. Socio-economic disadvantage, in addition to operating through a number of unhealthy behaviors and chronic diseases (Misra, O'Campo, & Strobino, 2001), also leads to an accumulation of chronic stressors and stress that may synergistically increase the risk of PTB (Berkowitz & Kasl, 1983; Canadian Institute of Child Health, 1993; Kramer et al., 2000; Kramer, Goulet, Lydon, Sequin, McNamara, & Dassa, 2001; Norbeck & Tilden, 1983) and LBW (Kogan, 1995; Longo, Kruse, Le Fevre, Schramm, Stockbauer, & Howell, 1999).

There is research evidence that SES exerts influence on other risk factors. More specifically, researchers have suggested that low SES may be a social cause of health-inhibiting behaviors such as smoking (Dow-Clarke, McAlder, & Hessel, 1994; Stewart, Gillis, Brosky, Johnston, Kirkland, Leigh, et al., 1996), poor nutrition (Hickey, McNeal, Menefee, & Ivet, 1997), increased susceptibility to genital tract infections (Fiscella, 1996; Kramer et al., 2001), inadequate housing (Kramer et al., 2000; National Council of Welfare, 1997), and poor working conditions (Chance & Walker, 1998). Many of these risk behaviors may increase a woman's risk of pregnancy complications and adverse birth outcomes. Investigators have also noted that low-income women, who are at risk for delivering a LBW or preterm infant, often experience barriers to prenatal care (e.g., transportation, child care, work flexibility) (Aved, Irwin, Cummings, & Findeisen, 1993; Beckman, Buford, & Witt, 2000; Lia-Holberg, Rods, Skovolt, Oberg, Berg, & Mullett et al., 1990; Stout, 1997), and often underutilize preventive health programs such as prenatal education classes (Edmonton Board of Health, 1994) and prenatal care (Cook, Selig, Wedge, & Gohn-Baube, 1999; Katz, Armstrong, & LoGerfo; 1994; Loveland Cook, Selig, Wedge, & Gohn-Baube, 1999; Melnikow, Alemango, Rottman, & Zyzanski, 1991; Mustard & Roos, 1994; Nothnagle, Marchi, Egerter, & Braveman, 2000; Perloff & Jafee, 1999; Poland, 1991).

Whether prenatal care would mitigate or decrease any of the risk factors for LBW or prematurity among low-income women is uncertain. Mustard and Roos (1994), in a study of 12,646 women in Winnipeg Canada, assessed the effectiveness of prenatal care on LBW across groups of women with different maternal socio-economic characteristics. They found that lower utilization of prenatal care by poorer women accounted for only a small difference in the mean birth weight (140 grams), and that the impact of pregnancy complications on birth weight was not substantially mediated by routine prenatal care utilization. In addition, O'Campo, Xue, Cheng-Weng and O'Brien-Caughy (1997) found that even when prenatal care was initiated early, the effects of early prenatal care were diminished for those women from neighbourhoods where unemployment was high. These results suggest that poor women may experience risk conditions that are beyond the reach of prenatal care alone. In other words, the social risks faced by women within the poor neighbourhoods may be such that the protective effect of prenatal care initiation and utilization diminishes.

SES has usually been operationally defined in the LBW and PTB research literature using individual level data such as income, occupational status, education, or having health insurance (Adler, Boyce, Chesney, Folkman, & Syme, 1993; Kaplan, 1996; Kogan, 1995; Lang et al., 1996; Meis et al., 1995). Only a few studies have used aggregate level socio-economic data (e.g., average family income, unemployment rate, percentage of immigrant population, percentage of single parent families) that considers the socio-economic profile of the mother's neighbourhood (Crosse, Adler, Ostbye, & Campbell, 1997; Kreiger, 1992; Kreiger, Williams, & Moss, 1997; O'Campo et al., 1997). Models utilizing individual level data have been able to explain only a small proportion of the effect of SES on LBW. O'Campo et al. (1997) recommended that investigations move beyond individual level data to consider models that include aggregate level census-based socio-economic risk factors (e.g., census family income; percentage population with less than grade 9 education, per capita crime rates) because of the known relationships between social inequality, environmental stressors, and health status, including poor pregnancy outcomes. Measurement of SES at the aggregate level turns the focus away from the individual to consider the environment in which people live, how the environment varies by social class, and how it impacts health behavior and adverse health outcomes such as LBW (Kreiger; O'Campo et al.).

Several investigators have explored whether census-based measures of SES can explain variations in LBW (Crosse et al., 1997; Kreiger, 1992; Luginaah, Lee, Abernathy, Sheehan, & Webster, 1999; O'Campo et al., 1997; Pearl, Braveman, & Abrams, 2001; Spencer, Bambang, Logan, & Gill, 1999) and PTB (Kaufman, Dole, Savitz, & Herring, 2003). O'Campo et al. in an effort to understand more about the mechanism(s) by which socio-economic risks are translated into LBW, conducted a study to determine if neighbourhood level socio-economic variables were related to LBW. Using computerized birth certificate information (i.e., birth weight, maternal age, education, health insurance status, trimester of prenatal care initiation) and census tract data (i.e., unemployment rate, rate of housing violations, per capita crime rate, average wealth, per capita income, ratio of homeowners to renters), these researchers completed a multilevel analysis to determine if individual level risk factors behaved differently depending on the characteristics of the neighbourhood in which a woman resided. They found that census-based per capita income was weakly associated with an increased risk of LBW (OR=1.11; CI: 1.02-1.22). Moreover, census-based socio-economic indicators, specifically unemployment, per capita income, and Medicaid health insurance, modified the association between the individual level factors and the risk of LBW. As unemployment in the area increased, the protective effect of early initiation of prenatal care diminished. In addition, the investigators reported that the increased risk of LBW for women with low levels of education (individual level factor) was stronger in high crime than in low crime neighbourhoods.

Census-based socio-economic factors have also been utilized to determine their role in explaining inter-area variations in LBW rates (Crosse et al., 1997; Lund, Modvig, Hilden, Rosdahl, Kure, & Schmidt, 1999). Crosse et al. examined small area variations within the district of London, Ontario. Both LBW rates and the rates for socio-economic factors (low income, low education, unemployment, percentage of immigrants, and percentage of unwed mothers and teen mothers) were calculated for each census tract in London, Ontario. Rates of teen mothers, unemployment, low income and low education were predictive of LBW. However, when unwed motherhood was taken into account, unemployment, low education, and low income did not add a unique contribution to the prediction of LBW. In a second Ontario study, significant associations were found between the similar variables (i.e., low income, single parenthood, teen motherhood, low education) and the risk of adverse birth outcomes including perinatal death, early neonatal death (END), stillbirth, and birth weight (Luginaah et al., 1999). More specifically, low income (r=0.89, p < .05) and single parenthood (r=0.82, p < .05) were associated with END, low education was associated with LBW (r=0.86, p <. 05), and teen motherhood was associated with birth weights between 750-1499 grams (r=0.82, p < .05), and 1500-2499 grams (r=0.86, p <.05).

Although neighbourhood socio-economic studies have provided insight into the potential processes by which SES may result in the birth of a LBW infant, there were several limitations in these studies. Investigators did not adjust adequately for individual level data (i.e., maternal medical and obstetrical factors, nutritional status, and lifestyle factors) or test for possible interactions among these risk factors. In order to increase our understanding of area variations or geographical differences in LBW and PTB, investigators have recommended examination of the influence of a more complete list of individual maternal risk factors and aggregate socio-economic risk factors (O' Campo et al., 1997, Crosse et al., 1997). Moreover, such analyses may successfully unravel or explain the mechanism by which neighbourhood socio-economic risk factors contribute to adverse birth outcomes.

To my knowledge, there is little information on how aggregate level factors influence maternal and fetal well-being. Goldman (2001) suggests that communities with poverty, high unemployment, and crime affect the health of residents through destabilizing family structures, eroding support, and increasing stress. Gelberg, Gallagher, Anderson and Koegel (1997) further propose that seeking preventive care may be a low priority for women who live in distressed neighborhoods where the limited social and economic opportunities place burdens on women, increasing the number, type, and magnitude of concerns, they face each day.

Genetic and Constitutional Factors

Maternal Height. Kramer (1987) reviewed 79 research studies that examined the effects of maternal height on birth weight (grams), gestational duration (weeks), and the rates of preterm and intrauterine growth restricted births. Although he found that mean birth weight decreased 7.8 g/cm for women whose height was less than 158 cm, he concluded that there was no obvious biological mechanism by which height could affect birth weight or gestational age. Investigators who have examined the effect of maternal height on PTB have suggested that women of short stature (i.e., maternal height defined as less than 158 cm) are at an increased risk of delivering a preterm infant (Kramer et al., 1992; Kramer, Coates, Michoud, Dagenais, Hamilton, & Papageorgiou, 1995; Pickering & Deeks, 1991; Lang et al., 1996; Meis et al., 1995). However, other researchers have found no effect of maternal height on gestational age (Abrams et al., 1991; Wen et al., 1990), after controlling for maternal weight (Berkowitz & Papiernik, 1993). These equivocal findings may be due to differential preterm definitions such as idiopathic PTB, preterm labor, and medically indicated PTB.

Pre-pregnancy Weight. Several studies have found that low pre-pregnancy weight (less than or equal to 55 kg) was associated with an increased risk (ORs ranged from 1.75 to 3.0) of delivering a SGA infant (Clausson, Cnattingius, & Axelson, 1998; Kramer, 1987; Spinillo Capuzzo, Piazi, Nicola, Colonna, & Iasci, 1994a). Studies have also shown an association between PTB and low pre-pregnancy weight (Ancel et al., 1999; Abrams & Newman, 1991; Lang et al., 1996; Meis et al., 1995; Wen et al., 1990) and low BMI (defined as pre-pregnancy weight for height index of less than 20 kg/m^2) (Ancel et al., 1999; Berkowitz et al., 1998; Hickey et al., 1997; Kramer et al., 1995; Schieve, et al., 2000; Schieve, Cogswell, & Scanlon, 1999). Risk estimates for low pre-pregnancy weight or low BMI and PTB have ranged from 1.3 to 4.0. With respect to the association of PTB and high maternal weight (BMI 26 kg/m^2 or pre-pregnancy weight 75 kg or greater), investigators have reported both increased risks (ORs 1.5 to 2.0) (Lang et al., 1996; Ancel et al., 1999; Zeitlin et al., 2001) and no increased risk (Berkowitz et al., 1998; Wen et al., 1990).

Obstetrical Factors

Parity. A number of studies have investigated the relationship between parity and birth outcomes including PTB (Lang et al., 1996; Meis et al., 1995) and LBW (Chumnijaraki et al., 1992; Wessel et al., 1996). Kramer (1987), based on his review of 74 studies, reported that an infant's mean birth weight increased by about 43.3 grams with increasing parity. Furthermore, first babies were found on average to be 82.7 grams lighter than multiparous infants. The majority of studies that evaluated the effect of parity on birth weight have done so examining the relationship between parity and the risk of fetal growth restriction (i.e., SGA births) (Abrams & Newman, 1991; Zeitlin et al., 2001). The two studies that investigated the impact of parity on LBW (less than 2500 grams) reported an increased risk of LBW for nulliparous women (ORs ranged from 1.6-5.2) (Chumnijaraki et al., 1992; Wessel et al., 1996). However, these studies were not able to control for a number of confounders that could modify the relationship between parity and LBW (e.g., height, pre-pregnancy weight, smoking, alcohol consumption, street drug use).

Different findings also have been reported for the association between parity and PTB. Both Kramer (1987) and Berkowitz and Papiernik (1993) suggest that parity does not play a role in PTB. However, Meis et al. (1995) and Lang et al. (1996) report an increased risk for PTB for nulliparous women, odds ratios 1.3 (CI: 1.06-1.65) and 1.8 (CI: 1.4-2.2) respectively.

History of Previous PTB or Previous LBW Birth. A previous history of a PTB is one of the most important risk factors for a subsequent PTB (Kramer, 1987; Lang et al., 1996; Malloy, 1999; Wen et al., 1990). Estimates of the relative risk for a woman with a history of PTB have been reported to be approximately 3.0 (Berkowitz & Papiernik, 1993; Kramer, 1987; Wen et al., 1990). This risk for PTB also increases considerably with the number of previous LBW or preterm infants. Bakketeig, Jacobsen, and Hoffman (1979), in a study of the recurrence of preterm delivery, reported that the risk of PTB increased from 14.3% in the second pregnancy, if the first pregnancy was preterm, to 28.1% in the third pregnancy when the previous two births were preterm.

Spontaneous and Induced Abortion. Kramer (1987), from his meta-analysis and synthesis of studies that examined a history of previous spontaneous abortion or induced abortion on birth weight, concluded that spontaneous and induced abortions did not have an effect on birth weight. On the other hand, Basso, Olsen, and Christensen (1998) concluded that women having a live birth preceded by a spontaneous abortion had a higher risk (OR= 1.32; CI: 1.25-1.68) of having a LBW infant. This finding must be viewed with caution, however, because the investigators did not control for confounders such as lifestyle factors, parity, infection, and pre-pregnancy weight (Kramer, 1987). Michielutte et al. (1992) also found an increased risk (OR= 1.42; CI: 1.17-1.72) for LBW with 2 or more previous abortions in the first trimester. It is not clear from the study design if the abortions were spontaneous or induced. From the evidence presented above, it is apparent that the association between previous abortion and LBW may be a result of the timing, type, or number of abortions.

The findings with respect to previous spontaneous abortion and the increased risk for PTB are inconclusive. Although several investigators suggest that there is an increased risk for PTB (Berkowitz and Papiernik, 1993; Kramer, 1987; Lang et al., 1996; Ancel et al., 1999) other investigators have not reported an increased risk (Abrams et al., 1989; Zeitlin et al., 2001). Investigators reporting a risk have suggested that the risk estimates for PTB vary according to the number of previous spontaneous abortions. The risk for PTB has been found to increase after 2 or more spontaneous abortions, with the risk estimates ranging from 1.5 to 3.3 (Lang et al., 1996; Berkowitz & Papiernik, 1993). Kramer suggested that the use of dilatation and curettage to remove placental tissue after spontaneous abortion could result in cervical incompetence and thus increase the risk of preterm delivery in subsequent pregnancies.

The effect of induced abortion on PTB and LBW has also been studied. Kramer (1987) and Berkowitz and Papiernik (1993) found no evidence in the studies they reviewed to suggest that there was an association between induced abortions and PTB. Lang et al. (1996) did report an increased risk for preterm labor, with the risk increasing from 1.9 for 2 abortions to 3.3 for women who had 3 or more induced abortions. Chuminijarakij et al. (1996) found that women who had 2 or more induced abortions had a 2-fold risk of LBW (RR=2.16; CI: 1.13-4.13). These findings suggest that the increased risk for PTB and LBW arising from induced abortions may be a function of the number of induced abortions and cervical incompetence.

History of Prior Stillbirth or Neonatal Death. Kramer (1987) in his synthesis of the literature found that a history of stillbirth or previous neonatal death had an impact on gestational duration, birth weight, and IUGR. Other investigators have also reported an increased risk of PTB for women who have had a previous stillbirth (Lang et al., 1996; Meis et al., 1995; & Robson, Chan, Keane, & Luke, 2001). Risk estimates for these studies range from 1.8 to 4.7. In contrast to Kramer's (1987) and Sanjose and Roma's (1991) findings of an increased risk (2-3 fold) for PTB for women who have had a previous neonatal death, Wessel et al. (1996) did not report an increased risk for PTB for women who had a previous neonatal death. Kramer (1987) suggests that many of the studies that have examined the effect of prior stillbirth or neonatal death on gestational age and IUGR have not separated the effect of prior stillbirth or neonatal death from that of prior prematurity, LBW, and spontaneous abortion.

Prenatal Care and Prenatal Education Classes

Prenatal care is accepted as a key component of preventive care for pregnant women. It is assumed that early and regular prenatal care will have a beneficial effect on pregnancy by identifying women "at risk" of having an adverse outcome, providing treatment for current pathological conditions, ensuring the diagnosis and treatment of complications, and providing health education for mothers (Mustard & Roos, 1994). Although many beneficial effects on maternal and perinatal health have been attributed to antenatal care, to date there is no conclusive evidence that prenatal care is effective in the prevention of LBW or PTB (Bergsjo & Villar, 1997; Frick & Lantz, 1999; Klermann, Ramey, Goldenberg, & Marbury, 2001; Kramer, 1997; Mustard & Roos, 1994).

Prenatal Visits. Most studies examining the impact of prenatal care on LBW and PTB have assessed adequacy of care according to the timing of prenatal care visits and the number of prenatal visits (Barros, Tavares, & Rodrigues, 1996; Dejardines & Hardwick, 1999; Lang et al., 1996). Few studies have examined the specific content of prenatal care visits (Sable & Herman, 1997).

Very few investigators have examined simultaneously the effect of a more complete list of risk factors in addition to inadequate prenatal care on poor birth outcomes. Furthermore, the definitions of adequacy of care and birth outcome also differed across the studies. Two research teams that attempted to be more inclusive in the number of risk factors examined and that controlled for appropriate confounders were Lang et al. (1996) and Kogan, Alexander, Kotelchuk, Nagey, and Jack (1994). Kogan et al. examined the relationship between content of care (e.g., health advice regarding smoking, drug use, alcohol, diet and medical procedures), prenatal care utilization (e.g., number of visits and initiation of care), and LBW. The study sample consisted of 9,394 women who delivered a liveborn infant. After controlling for socio-demographic factors (age, marital status, race, education, income, employment), medical conditions (hypertension), obstetrical history (parity, previous adverse birth outcomes), and lifestyle factors (smoking), women who reported not receiving all types of advice (diet, alcohol and drug use, smoking, proper weight gain, breastfeeding) were 38% more likely (OR=1.38; CI: 1.18-1.60) to have a LBW infant. Furthermore, women who did not initiate visits within an appropriate time frame and women who had an inadequate number of visits each had a 2-fold increased risk of delivering a LBW infant. Similarly, in Lang et al.'s study of 23 potential risk factors, late prenatal care (first prenatal visit occurring after the first trimester) was associated with an increased risk of preterm labor (OR=1.8, CI: 1.5-2.3).

In contrast to findings that suggest that late initiation of prenatal care, inadequate content of care, and inadequate number of prenatal visits negatively influences birth outcomes such as PTB and LBW, study findings also indicate that women who have initiated care by the 4th month and women who have an increased number of visits also have poor birth outcomes (Collins, Wall & David, 1997). These women are likely 'at risk' because of medical problems or pregnancy complications and thus required additional medical care. The empirical evidence supporting the association between prenatal care and reduced rates of prematurity and/or LBW is equivocal. There is conflicting evidence on whether or not standard prenatal care actually improves birth outcomes (Alexander & Korenbrot, 1995; Frick & Lantz, 1999; Klermann et al., 2001).

As a result of health care reform, cost constraints, and the lack of evidence to suggest that more prenatal visits result in better pregnancy outcomes, several investigators have challenged the cost effectiveness of the existing structure of prenatal care (i.e., frequency, timing, and content of care), indicating that more visits do not necessarily equate to better outcomes (Walker, McCully, & Vest, 2001; Villar, Carroli, Khan – Neelfour, Piaggo, & Gulmezoglu, 2002). Villar, Ba'ageel, Piaggo, Lumbigano, Belizan and Farnot (2001) suggest that prenatal care that endorses fewer goal directed visits (5-8 visits) can be as effective as standard models of care that recommend a greater number of visits (12-14 visits). Carroli, Villar, Paiggo, Khan-Neelfour, Gulmezoglu, and Mugford (2001) completed a systematic review to assess the effect of two different models of care on pregnancy outcomes such as LBW, PTB, urinary tract infection, and pre-eclampsia. A new goal oriented model of prenatal care (i.e., reduced antenatal visits with recommendations regarding types of preventive care and screening) was compared with the standard model of prenatal care. Of the trials reviewed, the number of visits in the new model of care ranged from 4-9, whereas in the standard model of care the number of visits was 13-14 visits. The results of this systematic review and meta-analysis suggest that there was no clinically-differential effect of the reduced visits when the results were pooled for a number of outcomes, including pre-eclampsia (OR=0.91; CI: 0.66-1.26), urinary tract infection (OR=0.93; CI: 0.79-1.10), postpartum anemia (OR= 1.01; CI: not provided), maternal mortality (OR= 0.91; CI: 0.55-1.51), and LBW (OR=1.04; CI: 0.93-1.17).

The results of this systematic review suggest that prenatal care can be provided with fewer visits, without any clinically important increase in the risk of adverse outcomes. Although these findings appear positive, it is important to note that the proportional reduction in the number of visits in the trials in more developed countries was actually very small. An absolute difference of three to four antenatal care visits in more developed countries where the norm is 11-14 visits is likely to be of little clinical significance (Carroli et al., 2001). If goal-directed counselling and screening is in place regardless of the number of visits, then the expected outcomes may be a result of the content of care in contrast to the number of antenatal visits. To understand if prenatal care does influence birth outcomes, future studies must consider both the quality and content of care received as well as the number of visits, while controlling for relevant confounders (Berglund & Lindmark, 1998; Desjardins & Hardwick, 1999; Goss, Lee, Koshar, Heilemann, & Stinson, 1997; Muender, Moore, Chen, & Sevick, 2000; Sable & Herman, 1997).

In summary, the findings with respect to the association between prenatal care and PTB and LBW remain equivocal (Blondel & Marshall, 1998; Kogan, et al., 1994; Melnikow et al., 1991; Poland, 1991). This may be due to methodological problems such as selection bias, inadequate control of confounders, and different definitions of the "adequacy" of prenatal care (i.e., content of care, quality of care, initiation of care, number of prenatal visits).

Prenatal Education. Prenatal classes are thought to provide a second opportunity for pregnant mothers and their support system to receive both health promotion and prevention information that may indirectly influence birth outcomes (Davies, Stewart, Sprague, Niday, Nimrod, & Dulberg, 1998). However, because prenatal class attendance is often a function of maternal age, parity, socio-economic status, and availability of classes, these opportunities are often missed (Anderson-Beckmann, Buford, & Will, 2000). Existing childbirth education research has been limited to examination of obstetric outcomes such as pain management, coping strategies, and increased use of forceps or vacuum assistance at delivery, and has not focused on the impact on birth outcomes (Sturrock & Johnson, 1990; Lynch & Young, 1997). In a recent Canadian survey of childbirth educators and practitioners about PTB prevention, it was found that 76% of the educators provided written material about PTB for women; however, limited time was spent reinforcing and discussing lifestyle issues and education about PTB (Davies et al., 1998). Several investigators have suggested that existing childbirth curricula should be examined not only for their content but also for the utilization of outcome measures related to maternal and infant health (Enkin, 1990; Lynch & Young, 1997).

Lifestyle Factors

Key maternal lifestyle factors that have been examined in the research literature in terms of their impact on birth weight and gestational age include smoking, alcohol consumption, and street drug use. The following literature review includes studies that provide information about the postulated mechanisms by which lifestyle behaviors affect the mother and unborn infant (Froom, Melamed, & Benbassat, 1998, Mathews, Yudkin, Smith, & Neil, 2000; Shah & Bracken, 2000); studies regarding the prevalence of PTB and LBW among women who engage in smoking, alcohol consumption, and street drug use (Cnattingius, 1997; Cnattingius, Mills, Yuen, Eriksson, & Salonen, 1997; Monica & Lilja, 1995; Savitz, Dole, Terry, Zhou, & Thorp, 2001); studies that report an increased prevalence of pregnancy complications among women who engage in specific lifestyle behaviors (Ananth, Demissie, Smulian, & Vintzileous, 2001; Odendaal, Schie, & de jeu, 2001); and studies that have examined whether the increased risks for PTB and LBW persist when both lifestyle and lifestyle-related confounders are examined concurrently (Kryklund-Blomberg & Cnattingius, 1998; Wisborg, Henriksen, Hedegard, & Secher, 1996; Zeitlin et al., 2000).

Smoking. Examinations of the effects of smoking on PTB and LBW have included investigations where active smoking and environmental tobacco smoke were considered (Lang et al., 1996; Meis et al., 1995; Windham, Hopkins, Fennester & Swan, 2000). It has been postulated that smoking affects the fetus by reducing fetal growth and birth weight through mechanisms such as inadequate maternal nutrition and poor maternal weight gain (Froom et al., 1998; Groff, Mullen, Mongoven, & Burau, 1997; Nandi & Nelson, 1992; Secker-Walker, Vacek, Flynn, & Mead, 1998; Mathews et al., 2000; Zaren, Lindmark, & Berjsjo, 1997; Zaren, Lindmark, & Bakketig, 2000) and by directly decreasing levels of maternal estrogen that acts as a fetal growth hormone (Lambers & Clark, 1996; Shah & Bracken, 2000). It has also been hypothesized that smoking results in PTB by initiating preterm labor through vasoconstriction actions in the placenta, which in turn may activate the stress pathways of the fetal placental axis via corticotrophin releasing hormone (Challis & Gibb, 1996; Gibb & Challis, 1998;

Chemlow, 1996; Salafia & Schiverick, 1999), and by increasing the likelihood of pregnancy complications such as placenta previa, placental abruption, and preterm premature rupture of membranes (Ananth, Demissie, Smulian, & Vintzileos, 2001; Kyrkland-Blomberg & Cnattingius, 1998; Odendaal, Schie, & de Jeu, 2001; Wong & Bauman, 1997). Both Kyrklund-Blomberg and Cnattingius (1998) and Wong and Bauman (1997) reported that the rates of abruptio placenta, placenta previa, and preterm rupture of membranes were greater for smokers than for non-smokers, and that there was a dose response effect: the rates of these pregnancy complications increased with the amount smoked. Odds ratios for smoking and placenta previa and for smoking and abruptio placenta, have ranged from 1.9 to 2.6 and 1.4 to 2.0 respectively (Cnattingius, 1997; Cnattingius et al., 1997; Monica & Lilja, 1995).

Horta, Victora, Memezes, Halpen and Barros (1997) and Kramer (1998) suggest that the effect of maternal smoking on LBW is significant, influencing both gestational duration and intrauterine growth; however, the effect on growth is more significant. These negative growth effects have been documented in terms of crown-heel length (Ohmi, Hirooka, & Mochizuki, 2002; Roquer, Figueras, & Jimenez, 1994; Zaren, Lindmark, & Berjsgo, 1997), head circumference (Lindley, Becker, Gray, & Herman, 2000), and infant proportionality indicators such as brain body weight ratios (Lindley et al., 2000) and ponderal index (Lindley, Gray, & Herman, 2000). It is also well established that cigarette smoking reduces the mean birth weight, with decreases in birth weight ranging from 149 grams to 300 grams (Hamilton, 2001; Kramer, 1987; Perkins, Belcher, & Livsey, 1997). In eight studies where LBW was defined as less than 2500 grams, women who smoked had approximately a 2-fold risk of LBW (Abel, 1997; Cnattingius, Forman, Berendees, Graubard, & Isolato, 1993; Dollberg, Seidman, Armon, Stevenson, & Gale, 1996; Horta et al., 1997; Moore & Zaccaro, 2000; Tough et al., 1999; Windham et al., 2000). However, only three of the eight studies controlled adequately for the majority of relevant confounders (i.e., age, parity, socio-economic status, pre-pregnancy height and weight) identified by Kramer (1987).

The findings regarding the association between cigarette smoking and PTB are inconsistent. Findings are dependent on how the birth outcome is defined, the number of cigarettes smoked, and the covariates considered (Ferraz, Gray, & Cunha 1990; Kyrklund- Blomberg & Cnattingius, 1998; Moore & Zaccaro, 2000; Wong & Bauman, 1997). Several investigators who controlled for confounders such as SES, height, weight, and parity have reported increased risks (OR= 1.2-3.0) for PTB (Kryklund-Blomberg & Cnattingius, 1998; Meis et al., 1995; Wisborg et al., 1996; Zeitlin et al., 2001). In the studies where additional confounders such as street drug use and other risk factors for PTB were controlled, the risk associated with maternal smoking was inconsistent (Abrams et al., 1989; Lang et al., 1996; Petridou, Salvanos, Skalidou, Dessypris, & Moustaki, 2001).

Lang et al. (1996) in a study of 23 risk factors on PTB did not find an increased risk of preterm labor with maternal smoking; however, there was an increased risk for SGA births (OR=2.2; CI: 1.9-2.5). In other studies, both Meis et al. (1995) and Zeitlin et al. (2001) found that women who smoked were not at an increased risk for medically indicated PTB. However, women who smoked were more likely to have a spontaneous PTB. Meis et al. found that an increased risk (OR=1.33; CI: 1.12-1.59) only existed for women who smoked more than 10 cigarettes a day, whereas Zeitlin et al. found that the risk for spontaneous PTB persisted regardless of the number of cigarettes (OR=1.29, CI: not reported).

Considerable interest has been voiced about the possible effects of passive smoking on LBW and PTB (Ahlborg & Bodin, 1991; Ahluwalia, Grummer- Strawn, & Scanlon, 1997; Chen & Petitti, 1995; Fortier, Brisson, & Marcoux, 1994). The literature on the association between environmental tobacco smoke (ETS) and LBW and PTB is equivocal. While Fortier et al. and Ahluwalia et al. found a slight association (OR =1.39; CI not reported) between ETS and LBW, other investigators have not reported an association (Ahlborg & Bodin, 1991). Findings for these studies must be interpreted with caution given the self-report method used to measure actual exposure, and the lack of control for confounders such as age, socio-economic status, and pre-pregnancy weight.

In summary, existing evidence suggests that women who smoke are at increased risk for delivering a LBW infant, and the effect of smoking on PTB may differ based on the PTB subtype (i.e., spontaneous preterm labor versus medically indicated PTBs).

Alcohol Consumption. Kramer (1987) in his review of 35 studies suggests that higher doses of alcohol have adverse effects on the growth of the fetus (i.e., a reduction in birth weight of 155 grams for women who have 2 or more drinks a day). Windham, Fennester, Hopkins, and Swan (1995) and Bada, Das, Bauer, Shankaran, Lester, Wright, Verter, Smeriglo, Finnegan, and Maza (2002) also reported decreases in birth weight with moderate to heavy alcohol consumption--decreases of 143 grams and 113 grams respectively. In studies examining the effect of alcohol consumption on LBW (less than 2500 grams), Shiono, Klebanoff, Nugent, Cotch, Wilkins, Rollins, Carey, and Behrman (1995) did not find an increased risk, whereas Windham et al. (1995) did find an increased risk (OR= 2.6; CI: 1.2-5.7) for LBW for women who consumed a moderate amount of alcohol (i.e., 3 or more drinks a week). In regard to PTB, Kramer (1987) suggests that the effects of alcohol on gestational duration and prematurity are both conflicting and unconvincing. Kesmodel, Olsen, and Secher (2000) reported that women who had 10-14 drinks/week had a 3-fold risk (RR=3.41; CI: 1.71-6.81) of PTB. However, Wen et al. (1990), Windham et al. (1995), and Shiono et al., (1995) all reported that women who consumed alcohol during pregnancy were not at increased risk for PTB. The inconsistent findings about maternal alcohol consumption and poor birth outcomes (i.e., LBW and PTB) may reflect differences in study methods including measurement of alcohol consumption, the ability to control for potential confounders, and sample size.

Street Drug Use. The evidence with respect to street drug use and an increased risk for LBW and PTB is inconsistent (Berkowitz & Papiernik, 1993). Both the early work of Kramer (1987) and a more recent meta-analysis to estimate the effect of maternal marijuana use on birth weight can be used to assert that there is inadequate evidence that marijuana use results in the birth of a LBW infant (English, Hulse, Milne, Holman, & Bower, 1997). In comparison to the studies reviewed by Kramer (1987) and Berkowitz and Papiernik (1993), which suggest that there is little evidence that marijuana affects gestational duration, Lang et al. (1996) reported an increased risk (OR=1.9; CI: 1.2-2.8) for preterm labor in marijuana users.

In contrast to marijuana use, cocaine use has been associated with a 2 to 3-fold increased risk of preterm deliveries (Kramer, 1997; Dinsmoor, Irons, & Christmas, 1994; Shiono, Klebanoff, Nugent, Cotch, Wilkins et al., 1995). In a meta-analysis of five studies of prenatal cocaine exposure, it was reported that there was an increased risk of LBW infants (pooled relative risk estimate 2.15; CI: 1.75-2.64). Furthermore, this risk increased with heavier cocaine use (pooled relative risk estimate 4.42; CI: 2.24-8.71) (English et al., 1997). Cocaine use has also been associated with an increased risk of pregnancy-related complications (e.g., premature rupture of membranes, placental abruption, and other infectious processes) that are known to result in a PTB (Chomitz, Leiberman, & Cheung, 1992; Dinsmoor, Irons, & Christmas, 1994; Lindeberg, Alexander, Gendrop, Nencioli, & Williams, 1991; Williams, Mittendorf, Lieberman, Monson, Schoenbaum, & Genest, 1991). This finding would suggest that cocaine may exert an indirect effect on birth weight and gestational age through increasing a woman's risk of pregnancy complications and early delivery (i.e., medically induced PTB).

Pre-existing Medical Diseases

Delay in the growth and development of the fetus has been associated with maternal pre-existing medical diseases and pregnancy complications that have been grouped in terms of their impact on the placenta, the pregnant women herself, the fetus, or some combination of these (Kramer, 1997). Understanding the mechanisms by which these medical factors interplay with other risk factors to result in PTB is important to reducing the overall LBW rate.

A number of maternal medical conditions such as chronic hypertension (Samadi & Mayberry, 1998) asthma (Alexander et al., 1998; Liu, Wen, Demissie, Marcoux, & Kramer, 2000; Tan & Thomson, 2000; Sorensen, Dempsey, Xiao, Frederisk, Luthy, & William, 2003), Crohn's disease (Norgard, Fonager, Sorensen, & Olsen, 2000), and diabetes mellitus (Coetzee & Levitt, 2000; Feig & Palda, 2002) have been associated with PTB. It has been estimated that women with pre-existing conditions such as chronic hypertension are 2 to 3 times more likely to deliver a preterm infant (Samadi & Mayberry, 1998). It is thought that these medical conditions either directly or indirectly impede delivery of nutrients or oxygen to the uteroplacental circulation. Consequently, the fetus cannot maintain normal growth and development. In addition to the effects on the fetus, these medical conditions may pose problems to the mother, which necessitates early delivery. Berkowitz and Papiernik (1993) conclude that the reported risk estimates of maternal chronic disorders on PTB must be interpreted with caution because the observed association may be due to early medical intervention.

In addition to chronic medical conditions, common episodic illnesses such as nausea, vomiting, diarrhea, anorexia, fever, urinary tract infections, and upper respiratory infections could potentially affect intrauterine growth or gestational duration. These conditions may deplete maternal energy and nutrition, or transmit infections to the placenta or amniotic fluid. For example, it is proposed that preterm labor and/or premature rupture of membranes results from the actions of pro-inflammatory cytokines secreted as part of the fetal or maternal host response to microbial infection (Dudley, 1997).

A pregnancy complication that is drawing considerable attention because of its role in LBW is genital tract infection (Gibb & Challis, 1998; Kimberlin & Andrews, 1998; Paige, Augustyn, Adih, Witter, & Chang, 1998; Stewart, 1998a). Infection of the upper genital tract is linked to spontaneous preterm delivery. More specifically, bacterial vaginosis (BV) associated organisms have been isolated from the upper genital tract of women with preterm labor and PTB (Hillier, Nugent, Eschenbach, Krohn, Gibbs, & Martine 1995, Goldenberg, Hauth, & Andrews, 2000; Flynn, Helwig, & Meurer, 1999). The relative risks for PTB associated with BV range from 1.4 to 3.1 (Eshenbach, Gravett, Chen, Hoyme, & Hoyme, 1994;Rauh, Culhane, & Hogan, 2000).

Flynn et al. (1999) conducted a meta-analysis to determine the magnitude of risk conferred by BV on preterm delivery, LBW, preterm premature rupture of membranes and preterm labor. Of the 233 studies reviewed, 39 studies were identified for possible inclusion (27 observational studies and 12 trials). The pooled data representing more than 17,000 patients demonstrated that women with BV were more likely to deliver a preterm infant (OR=1.85; CI: 1.62-2.11) or an infant weighing less than 2500 grams (OR =1.57; CI: 1.32-1.87). For preterm premature rupture of membranes and preterm onset of labor, the resulting ORs were 1.83 (CI: 1.39-2.44) and 2.19 (CI: 1.72-2.76), respectively. The consistency of these and earlier findings, the magnitude of relative risk, and the biological plausibility of bacterial vaginosis leading to PTB, all strongly suggest that bacterial vaginosis plays a significant role in preterm LBW. As a result of these findings, PTB researchers are redirecting their focus to examine the molecular and physiological mechanisms by which the presence of upper genital tract infections result in PTB (Gibb & Challis, 1998; Goepfert & Goldenberg, 1995; Goldenberg, Iams, Mercer, Meis, Moawad, & Cooper, 1996; MacDermott, 1995; Moore, 1999; Subtil, Denoit, Le Gouff, Husson, Trivier, & Puech, 2002).

Maternal Nutrition

A number of investigators have evaluated the association between nutritional status and birth weight and PTB (Kramer, 1998; Neggers, Goldenberg, Tamura, Johnston, Cliver & Hoffman, 1997; Siega-Riz, Adair, & Hobel, 1998). The nutritional studies that have been evaluated with respect to birth outcomes have primarily focused on weight gain during pregnancy and anemia during pregnancy.

Weight Gain During Pregnancy. The majority of studies linking maternal weight gain with LBW focused on intrauterine growth restriction (i.e., preterm and term small for gestational age births) (Clausson et al., 1998; Smith, Smith, Mc Nay, & Flemming, 1998; Spinillo et al., 1994a; Spinillo, Capuzzo, Nicola, Colanna, Egbe, & Zara, 1994b; Strauss & Dietz, 1999) rather than LBW defined as less than 2500 grams. PTB studies have also differed in their definition of the birth outcome, examining the association between maternal weight gain and medically induced PTB (Meis et al., 1995) or spontaneous PTB (Berkowitz et al., 1998), or more globally as gestational age less than 37 weeks (Abrams & Newman, 1991; Wen et al., 1990). While these studies have reported an association between low weight gain and PTB, the ability to determine what biological mechanisms may be responsible for such associations has been limited.

Carmichael, Abrams, and Selvin (1997) suggest that maternal weight gain could be a marker for maternal infection, maternal nutritional status with regard to certain micronutrients such as iron, deposition of maternal fetal stores, or differences in the ability to deliver nutrients from the mother to the fetus.

In contrast to the conclusions of Berkowitz and Papiernik (1993) and Kramer (1987) that low pregnancy weight gain is either weakly associated or not associated with risk of prematurity, other investigators suggest that women with a low rate of weight gain (weight gain 0.40-0.50 lbs/week) have an increased risk for PTB (ORs for PTB ranged from 1.5-3.0) (Abrams et al., 1991; Lang et al., 1996; Wen et al., 1990). Carmichael et al.'s (1997) critical review of 13 studies examining the relationship between gestational weight gain and preterm delivery concur that an inadequate rate of weight gain is associated with an increased risk of PTB (approximately 50-100%).

A number of studies indicate that the risk for PTB varies according to both the BMI and pregnancy weight gain. de Haas et al. (1991) and Spinillo et al. (1998) found that women who had both a low pregnancy weight gain (< 0.37 kg-0.5 kg/week) and a low BMI (< 19.5 kg/ m^2) had a 200% and 240% increased risk of PTB, respectively. Hickey, Cliver, Mc Neal, Hoffman and Goldenberg (1995) found that women with a low BMI (< 19.8 kg/ m^2) and a low third trimester weight gain (< 0.38 kg/week) had an increased risk of spontaneous PTB. Schieve, Cogswell, Kelley, Scanlon, Perry, and Ferre (2000), in a study of 3,511 mother-infant pairs, reported that both women with an average BMI (19.8-26.0 kg/m^2) and low weight gain (0.5-1.5 lbs/week) and women with a low BMI (under 19.8 kg/m^2) and low weight gain (0.5-1.5 lbs/week) had an increased risk for PTB, OR equal to 3.6 (CI: 1.6-8.0) and 6.7 (CI: 1.1-40.6), respectively. Schieve, Cogswell, and Scanlon (1999) also reported significant risk differences of 6.5% for women with low weight gain and average BMI. These differences did not change with control for SES. Other confounders were not identified or controlled for in the study. It was also not clear if the births included or excluded medically induced PTB or women with other pre-existing diseases. Although studies have differed both in terms of how PTB was defined and how gestational weight gain was measured, recent evidence suggests that low maternal weight gain alone, as well as low maternal weight gain in combination with low BMI are associated with an increased risk for PTB.

Anemia During Pregnancy. Several potential biological mechanisms have been identified through which anemia or iron deficiency could affect pregnancy outcomes. Anemia (by causing hypoxia) and iron deficiency (by increasing serum norepinephrine concentrations) can induce both fetal and maternal stress, which stimulates the synthesis of corticotrophin-releasing hormone (CRH) (Allen, 2001). Elevated CRH concentration is a major risk factor for preterm labor, gestational hypertension, pre-eclampsia, and premature rupture of the membranes (Allen, 1997; Steer, 2000). Furthermore, CRH increases fetal cortisol production, which in turn may inhibit longitudinal growth of the fetus (Scholl & Reilly, 2000). Alternative mechanisms that also have been identified suggest that a) iron deficiency increases oxidative damage to the erythrocytes and the fetal-placental unit; and b) iron deficiency may increase the risk of maternal infections, which then stimulate the production of CRH (Allen, 2001).

Several investigators have studied the impact of anemia on fetal growth restriction and LBW. Scanlon, Yip, Schieve, and Cogswell (2000) and Scholl, Heidger, Fischer, and Schearer (1992) did not find an association between severe anemia (hemoglobin < 95g/L at 12 weeks gestation; serum ferritin <12 ug/L in the first trimester) and the birth of small for gestational age infants. Conversely, Bondevik, Lie, Ulstein, and Kvale (2001) reported that women with severe anemia (hematocrit ≤ 24 %) had a 2-fold risk (OR=2.4; CI: 1.01-5.8) of delivering a LBW infant. Although these studies were consistent as to when the anemia measure was taken, Bondevik et al. did not control for confounders such as smoking; consequently, this finding needs to be viewed with caution.

The findings with respect to anemia and PTB are also equivocal. Findings differ according to the definition of anemia (hematocrit, hemoglobin, serum ferritin) and /or definition of birth outcome (PTB defined as less than 37 weeks gestation, medically indicated PTB or spontaneous PTB). Scanlon et al. (2000) and Scholl et al. (1992) reported increased risk estimates of 1.68 (CI: 1.29-2.21) and 2.67 (CI: 1.13-6.30), respectively. Both investigators defined PTB as birth occurring before 37 weeks and anemia was determined in the first trimester. However, Scanlon et al. determined anemia by hemoglobin and Scholl et al. by serum ferritin levels. On the other hand, Meis et al. (1995) and Siega-Riz et al. (1998) did not report increased risks of preterm labor and preterm premature rupture of membranesin women with anemia. Meis et al. did not identify when the hemoglobin level was obtained; consequently, it is not known during what trimester the measure was taken. Hemoglobin levels taken in the third trimester may not discriminate between low hemoglobin caused by iron deficiency from that caused by plasma volume expansion (Allen, 2000).

Xiong, Beukens, Alexander, Demianczuk, and Wollast (2000), in a meta-analysis of 10 studies, found that anemia (defined as a hemoglobin of less than 10-11g/dL or a hematocrit less than 30-33%) in early pregnancy (< 20 weeks gestation) was associated with a slightly increased risk (pooled adjusted OR=1.2[illegible]; CI: 1.06-1.43) for PTB (< 37 weeks). However, this analysis included only eight studies, two of which were from developing countries and three in which the hemoglobin level was determined in late pregnancy (greater than 30 weeks gestation). Hemoglobin levels should be measured early in pregnancy, otherwise the effect is confounded with that of the expanded plasma volume that occurs in late pregnancy (Allen, 2000; Kramer, 1987).

Pregnancy Complications

There are a variety of different medical and obstetrical factors that have been linked to adverse outcomes such as LBW or PTB. Some of these factors can be diagnosed prior to pregnancy, such as maternal chronic illness and a history of poor obstetrical outcomes; other problems occur during pregnancy, such as gestational hypertension, gestational bleeding, and placental abnormalities.

Among the more important complications associated with preterm delivery and fetal growth restriction are placenta previa and abruptio placenta (Lang et al., 1996); antepartum bleeding (Ananth, Berkowitz, Savitz, & Lapiniski, 1999; Meis et al., 1995; Yang & Savitz, 2001; Zeitlin et al., 2001); gestational hypertension; and pre-eclampsia (Paruk & Moodley, 2000; Walker, 2001; Zeitlin et al., 2001). It is postulated that pregnancy complications are intermediate or intervening outcomes because other risk factors may have an impact on gestational duration or fetal growth indirectly through their effects on these complications (Abu-Heija, Al-Chaliabi, & El-Houbani, 1998; Lang et al., 1996). These complications can then in turn result in the reduction of nutrients to the fetus, play a role in the initiation of preterm labor, or lead to medically induced early delivery because either the mother or fetus is being adversely affected (Gibb & Challis, 1998).

Although investigators have differed in their definition of preterm delivery (i.e., preterm labor, preterm premature rupture of membranes, spontaneous PTB, PTB less than 37 weeks), the direction and magnitude of some of the complications are comparable across studies. Both Lang et al. (1996) and Meis et al. (1995) reported odds ratios of 2.7 (CI: 2.2-3.3) and 1.96 respectively (CI: 1.61-2.39) for bleeding and preterm labor and bleeding and spontaneous PTB. In contrast to Lang et al., Berkowitz et al. (1998) reported almost a 3-fold (OR=2.8; CI: 2.1-3.8) increased risk for preterm premature rupture of membranes related to antepartum bleeding and nearly a 4-fold risk for preterm labor related to antepartum bleeding (OR=3.7; CI: 2.5-5.5). Yang and Savitz (2001) after adjusting for maternal age, education, race, prenatal care, and smoking found that antepartum bleeding was strongly associated with PTB before 34 weeks (OR=2.2; CI: 1.3-3.8), and was notably stronger among women with severe bleeding in both pregnancy intervals.

Although multiple gestation births may not belong in the same category as other pregnancy complications, they are strongly associated with PTB (Berkowitz & Papiernik, 1993; Keith, Oleszczuk, & Keith, 2000). In addition, among those women who have multiple births, approximately 50% of the infants are both preterm and LBW (Joseph, 1998). Although multiple gestations account for only 2% of all pregnancies, they account for approximately 14% to 16% of all PTBs in Canada (Joseph, 1998). The largest increase in multiple births is in women over 30 years of age (Millar, Wadhera, & Nimrod, 1992). Current Canadian trends suggest that a substantial proportion of multiple births may be the result of delayed childbearing and the use of reproductive technologies.

For example, in Canada, the proportion of first births among mothers aged 30-34 years rose from 65% in 1974 to 86 % in 1994. For mothers aged 35 to 39, the percentage of first births was 25%, an increase from 13% in 1974 (Ford & Nault, 1996). In addition to the increase in first births to older women, an increase in the percentage of multiple births is also occurring. In Canada, multiple births increased from 1.9% in 1981-1983 to 2.1% in 1992-1994 to 2.3% in 1995 (Canadian Perinatal Surveillance System Steering Committee, 2000). In Alberta similar trends were observed; multiple births increased from 2.1% in 1991/92 to 2.5% in 1996/97 (Alberta Health and Wellness, 1999).

Current fertility trends, such as delayed childbearing (i.e., first births to women over 30 years of age), the availability and use of fertility enhancing technologies including in vitro fertilization (IVF), and the associated increase in multiple gestation births suggest that the rates of PTB will continue to increase (Joseph et al., 1998; McElraith & Wise, 1997). In a recent study by Tough, Newburn-Cook, Johnston, Svenson, Rose, and Belik (2002), the impact of delayed childbearing on population rate changes in LBW (less than 2500 grams), preterm delivery (less than 37 weeks gestation) and SGA births in Alberta between 1990 and 1996 was quantified. The results indicated that delayed childbearing was responsible for a substantial proportion of the population changes in LBW and preterm delivery; that is, 78% of the change in the LBW rate, and 36% of the change in the preterm delivery rate. Moreover, women aged 35 years and over were at a 20% to 40% increased risk of LBW and preterm delivery.

In Alberta, the rate of preterm multiple births also affects the LBW rate. Between 1996 and 1997 in Alberta, 56% of multiple birth newborns were LBW and 57% were preterm (Alberta Health and Wellness, 1999). Furthermore, the percentage of preterm multiple births and the percentage of multiple birth LBW babies in Alberta have increased over time. The percentage of preterm multiple births has increased from 45% in 1993/94 to 57% in 1996/97 and the percentage of multiple birth LBW babies has increased from 46% to 56% for the same time period (Alberta Health and Wellness).

Despite the trends of increased multiple births and their association with preterm LBW, multiple births represent only 16% of the Canadian LBW infant population

(Canadian Institute of Child Health, 1993) and 20% of the Alberta LBW infant population (Alberta Health and Wellness, 1999). Consequently, a reduction in the number of multiple births due to reproductive technology such as IVF would not substantively decrease the incidence of LBW.

Newborn Factor: Gestational Duration

Key determinants of LBW are duration of gestation and growth rate of the fetus (Kramer, 1987; Michielutte et al., 1992; Tough et al., 1999). Consequently, investigations have focused on examination of the independent (direct) effect of risk factors on: a) PTB (< 37 weeks gestation) and subtypes of PTB (i.e., preterm labor, preterm premature rupture of membranes, medically indicated PTB); b) intrauterine growth restriction (small for gestational age infants whose birth weight is less than the 10th percentile for gestational age using sex-specific criteria); or c) LBW (birth weight less than 2500 grams). No literature was found that examined whether the impact of risk factors on LBW operated indirectly through gestational age.

Summary of the Literature Review

A review of the risk factor literature suggests that: a) there are only a few studies that have examined a comprehensive number of risk factors for LBW and PTB while simultaneously controlling for known confounders; b) risk factor estimates may differ according to how investigators have defined both LBW (SGA versus birth weight less than 2500 grams) and PTB (i.e., preterm premature rupture of membranes, preterm labor; gestational duration less than 37 weeks); and, c) there is a need to further advance existing knowledge about LBW and PTB by attempting to understand how risk factors exert their effects directly and indirectly.

What then is our current understanding about the risk factors for PTB and LBW? What risk factors have confirmed associations with PTB or LBW? What findings are equivocal?

The findings for the effect of socio-demographic variables, such as maternal age and SES, on LBW and PTB are equivocal. Investigators who have controlled for age-dependent confounders have found both associations and no associations between maternal age (defined as maternal age less than 20 years and greater than or equal to 35 years of age) and PTB. With respect to SES, there were virtually no studies that have simultaneously examined the effect of aggregate level SES data and individual lifestyle, medical, and obstetrical risk factors on LBW and PTB. However, both Kramer's meta-analysis of individual SES measures and O'Campo et al.'s work examining specific aggregate SES measures and prenatal care would suggest that SES has an indirect effect on LBW, exerting its influence through nutrition, prenatal care, lifestyle (i.e, smoking, alcohol consumption, street drug use), medical problems, and anthropometric factors such as pre-pregnancy height and weight.

There is increasing evidence to suggest that women with a low BMI (combined index for pre-pregnancy height and weight) or a low pre-pregnancy weight are at increased risk for PTB. However, more evidence is required to confirm this relationship with both birth outcomes (LBW and PTB) and to determine if the equivocal findings for pre-pregnancy height can be refuted. This is especially important because more recent findings suggest an increased risk for PTB when BMI is used.

There has been minimal examination of the effect of maternal weight gain during pregnancy on LBW. However, the majority of the more recent PTB studies do suggest an increased risk for PTB for women who have a low gestational weight gain. In contrast to this nutritional indicator, the results regarding the effects of anemia during pregnancy on PTB remain equivocal.

A woman's obstetrical history does impact future pregnancy outcomes. A consistent finding in the research literature is the increased risk of PTB for women who have had a previous PTB. However, the findings regarding the effect of other obstetrical factors such as previous stillbirths and previous abortions remain equivocal.

Equivocal findings exist for prenatal care and several lifestyle factors. Investigators who have controlled for key confounders of prenatal care suggest that women with inadequate prenatal care are at increased risk for PTB (Lang et al., 1996) and LBW (Kogan et al., 1994). However, in clinical trials with goal directed content of care, birth outcomes do not differ for women with fewer prenatal visits, that is, there is no increased risk of LBW (Carroli et al., 2001).

Investigators who have controlled for key confounders associated with smoking reported both increased and decreased risks of PTB. It remains uncertain if these study differences are a result of how the birth outcome of PTB was defined (i.e., preterm labor, premature rupture of membranes, spontaneous PTB) or if the null findings indicate that the effect of smoking on PTB was indirect (i.e., through other risk factors). In contrast to the PTB findings, research data do support that women who smoke during pregnancy are at increased risk of delivering a LBW infant.

Congruent with earlier research, recent studies provide little evidence to support an increased risk of poor birth outcomes for women who smoke marijuana. On the other hand, there is evidence to suggest that the use of cocaine during pregnancy does increase the risk of delivering a preterm infant. It is difficult to determine from existing studies if cocaine use has an indirect effect on PTB operating through complications of pregnancy.

Lastly, pregnancy complications and specific pre-existing medical conditions have been shown to influence pregnancy and birth outcomes. These factors can adversely affect the health of the fetus and/ or mother necessitating medical intervention (i.e., induction or operative delivery) and the delivery of an infant preterm (less than 37 weeks gestation).

Many investigators have been limited in their ability to investigate concurrently the effect of multiple risk factors on PTB and LBW. Furthermore, there is limited research that has moved beyond examination of individual level data to determine if aggregate data such as neighbourhood SES in conjunction with individual data can provide a better explanatory model for LBW and PTB. Until research addresses individuals within the contexts in which they live and work, the relative importance of targeting individual or contextual factors to improve fetal well-being will remain unknown (O' Campo et al., 1997; Crosse et al., 1997). Information derived from research that examines both aggregate and individual level data would be important to public health practitioners and policy planners in determining where limited resources should be directed to improve population health (individual and/or macro level/contextual factors), as well as the specific intervention strategies needed to reduce the prevalence of preterm and term LBW outcomes (Moutquin, Bigirimana, Bernard, Hache, & Desmaris, 1997; Moutquin & LaLonde, 1998).

A Hypothetical Model of Potential Risk Factors for Low Birth Weight

The extensive review of the PTB and LBW risk factor literature was used to develop a hypothetical model that would provide direction for the modelling of the risk factors for LBW and PTB. Several factors influenced the development of the hypothetical model: a) consideration of the potential risk factors that were present prior to the index pregnancy (i.e., maternal age, genetic and constitutional factors such as height and weight, obstetrical history, socio-economic status); b) critical reflection on the distal and proximal causes and etiological pathways leading to LBW or preterm birth. For example, the effect of a distal factor such as young maternal age on LBW may be indirect and operate through more proximal factors such as lifestyle factors or prenatal care factors; c) examination of the various intuitive and logical relationships between different risk factors (i.e., prenatal visits and prenatal care may affect the status of pre-existing diseases, medical and nutrition problems during pregnancy, and pregnancy complications); and, d) deliberation about the multifaceted and interactive nature of these risk factors and their differential impact (i.e., older women are more likely to experience pregnancy complications, which in turn can result in a poor birth outcome).

The risk factors for LBW are depicted in Figure 1. This figure *only* represents the ordering of the risk factors (i.e., the order considers the distal and proximal relationship of the risk factors and the relationship between the risk factors and the birth outcome). The figure does not distinguish between direct and indirect effects of the risk factors on LBW. Individual risk factors were categorized into broader categories and examined in the model as a block. However, individual pregnancy complications and risk factor interactions were entered separately rather than as a block.

Maternal age is identified as the most distal risk factor in the model because it was thought that a number of the subsequent factors were a function of age (i.e., genetic and constitutional factors such as pre-pregnancy height and weight, a woman's obstetrical history, pre-existing chronic diseases, socio-economic status, medical problems during pregnancy, prenatal care, pregnancy complications, lifestyle factors). Thus, it was believed that the effect of maternal age was likely to be indirect. For example, younger women are more likely to be at risk for a LBW infant because they are less likely to have prenatal care and are more likely to engage in risk taking behaviors, such as smoking and illicit drug use.

Aggregate socio-economic status risk factors are located following maternal age because it was thought that the socio-economic environment was a function of maternal age and reflected the environment in which the mother lived. Furthermore, it was believed that socio-economic status may have an indirect effect on the mother's health and her health seeking behaviors.

The genetic and constitutional factors (pre-pregnancy height and weight) and the obstetrical history risk factors are the third and fourth blocks of variables presented in the model. These blocks of variables are positioned here because they are both a function of maternal age and are present prior to the index pregnancy. Prenatal classes and prenatal visits are factors that are specific to the index pregnancy and can potentially affect the health status of the woman and infant. Consequently, they are presented in the framework prior to those factors that may be influenced by prenatal care (i.e., lifestyle factors, such as smoking, alcohol consumption, and street drug use; pre-existing diseases; nutritional problems; medical problems; and pregnancy complications). Similarly, pre-existing diseases may affect nutrition and medical problems during pregnancy, or specific pregnancy complications. Consequently, these factors are situated prior to the pregnancy complications. Medical problems are also placed prior to pregnancy complications because it was hypothesized that these conditions (e.g., gestational diabetes, polyhydramnios or oligohydramnios) may result in either spontaneous PTB or a medically indicated PTB.

Pregnancy complications are positioned within the model prior to the birth outcomes because they are considered intervening factors in terms of the birth outcomes PTB and LBW. Pregnancy complications were entered in the model one at a time to determine if the effect of other risk factors on PTB and LBW was through pregnancy complications (indirect) or if the risk factors had a direct effect on the birth outcomes. Each complication was entered separately to determine both the direct and indirect effect of the pregnancy complication on the birth outcome. Lastly, gestational age is included in the model not only to examine the influence of the risk factors on gestational age but also to understand better whether the effects of the risk factors operate directly on birth weight or indirectly through gestational age. In this study, the gestational age of an infant was defined as an infant born prior to 37 weeks gestation. It was not was possible to consider the heterogeneity of LBW and to examine the indirect and direct effects of the study risk factors for preterm appropriate for gestational age infants, preterm small for gestational age infants, and term small for gestational age. This was due to the inadequate numbers of infants in each of these categories, the large number of risk factors being examined, as well as the low prevalence of some of the risk factors in these groups.

Figure 1. Hypothetical Model of Potential Risk Factors for Low Birth Weight

Maternal Age (younger & older women)
↓
Socio-economic Status (income, level of education, % single parents, % aboriginal population)
↓
Genetic and Constitutional Factors (pre-pregnancy height and weight)
↓
Obstetrical History (abortions, stillbirths, neonatal death, preterm birth, parity, SGA birth)
↓
Prenatal Care (prenatal visits, prenatal classes)
↓
Lifestyle Factors (smoking, alcohol consumption, street drug use)
↓
Pre-existing Diseases (heart disease, diabetes, chronic hypertension, renal, other medical diseases such as epilepsy, severe asthma, lupus, Crohn's)
↓
Nutritional Problems During Pregnancy (anemia, poor gestational weight gain)
↓
Medical Problems During Pregnancy (gestational diabetes, poly/olioghydramnios, acute medical problems such as urinary tract infection, acute asthma, thyrotoxicosis)
↓
Pregnancy Complication (gestational hypertension)
↓
Pregnancy Complication (gestational hypertension with proteinuria)
↓
Pregnancy Complication (bleeding less than 20 weeks)
↓
Pregnancy Complication (bleeding greater than or equal to 20 weeks)
↓
Pregnancy Complication (placenta previa)
↓
Gestational Age

↓

Low Birth Weight

Chapter 3 Methods

This chapter begins with a description of the study location, study design, study objectives, and study subjects. Next, descriptions of the data sources, data linkages, the assembled data file, and data transformations are provided. The chapter concludes with a detailed description of the data analysis and ethical considerations.

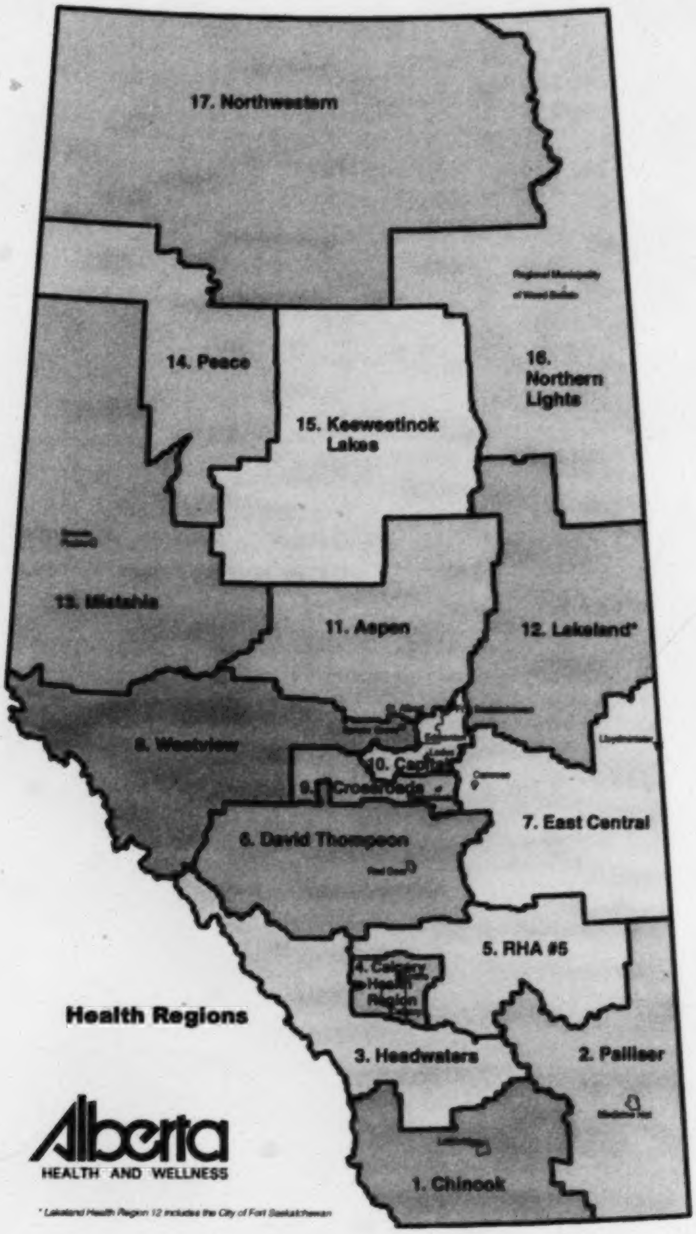
Study Location

The study location is the Capital Health Region in the province of Alberta, Canada. Alberta is a western Canadian province with a population of 2,696,082 people (Statistics Canada, 1997).

Capital Health is one of the two largest urban Regional Health Authorities (RHAs) within Alberta. Capital Health is one of Canada's largest integrated academic health regions, providing complete health services to 980,000 residents in the cities of Edmonton, Fort Saskatchewan, Spruce Grove, St Albert, and the counties of Leduc, Strathcona, Parkland, and Sturgeon (Statistics Canada, 1997). Within Capital Health there are 14 hospitals, 17 public health service centres, as well as long-term care facilities. Capital Health serves a total of 1.6 million people across central and northern Alberta, providing specialized services such as trauma and burn treatment, organ transplants, and high-risk obstetrical care. In addition to the services provided within the Capital Health region, core services of Capital Health are also provided to adjacent RHAs such as Aspen RHA (RHA 11), Lakeland RHA (RHA 12), Crossroads RHA (RHA 9), and East Central RHA (RHA 7). To facilitate service delivery within Capital Health, the region is subdivided into 17 Public Health Service Areas (PHSAs) defined by the Medical Officer of Health.

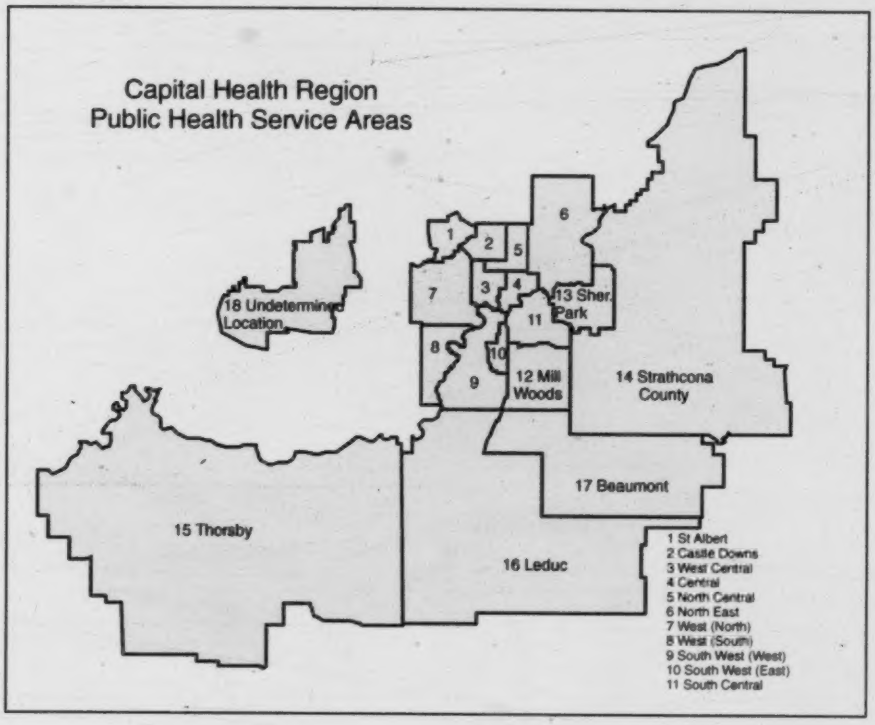
Figure 2 depicts the 17 RHAs in Alberta that were in place at the time of the study. These RHA boundaries were in force until 2003. Figure 3 depicts the 17 PHSAs in Capital Health and an undetermined PHSA called PHSA #18.

Figure 2. Map of Province of Alberta Regional Health Authorities


17. Northwestern
Regional Municipality of Wood Buffalo
14. Peace
16. Northern Lights
15. Keeweetinok Lakes
13. Mistahia
11. Aspen
12. Lakeland*
8. Westview
10. Capital
9. Crossroads
7. East Central
6. David Thompson
5. RHA #5
4. Calgary Health Region
3. Headwaters
2. Palliser
1. Chinook
Health Regions
Alberta
HEALTH AND WELLNESS

* Lakeland Health Region 12 includes the City of Fort Saskatchewan

Figure 3. Map of Public Health Service Areas Within Capital Health Region


Capital Health Region
Public Health Service Areas
1
2
5
6
7
3
4
13 Sher. Park
11
8
10
9
12 Mill Woods
14 Strathcona County
18 Undetermined Location
15 Thorsby
16 Leduc
17 Beaumont
1 St Albert
2 Castle Downs
3 West Central
4 Central
5 North Central
6 North East
7 West (North)
8 West (South)
9 South West (West)
10 South West (East)
11 South Central

Study Design

A population-based retrospective cohort design was used to address the research objectives.

Study Objectives

1) to examine the simultaneous impact of previously identified risk factors on birth weight (LBW) and gestational age (PTB);

2) to identify the direct and indirect effects of these risk factors on birth weight and gestational age; and

3) to illustrate and report the differences in the prevalences of risk factors within each of the PHSAs in CHA.

Study Subjects

The study subjects included all women who delivered a liveborn singleton infant and were residents of Capital Health between 1996 and 1998. Although multiple gestation births are a major risk factor for LBW and PTB, there is reason to believe that the mechanisms for PTB and LBW births for multiple and singletons are sufficiently different that including both in the same model would lead to confounding (Kramer, 1987; Lang et al., 1996). Similarly, different processes underlie infants with anomalies and stillbirths. Therefore, women who had a multiple gestation pregnancy, a stillbirth, or an infant with a major fetal anomaly were excluded from the study.

Data Sources

The study variables were contained in four databases: the Northern and Central Alberta Perinatal Audit Program Database (NCAPAPD), the Physician Notice of Birth (PNOB), the Alberta Vital Statistics Database, and the Statistics Canada 1996 Census Public Use Microdata Files.

Northern and Central Alberta Perinatal Audit Program Data

The NCAPAPD is an administrative perinatal database that contains a record for each birth in Northern and Central Alberta. Data are collected from health care facilities providing perinatal care throughout Health Authority Regions 6 to 17. Data are collected by participating hospitals using one of three methods: a) directly from the Provincial

Delivery Record (Part 1 and Part 2), b) a log book that is transcribed from the Provincial Delivery Record, or c) by electronic transfer of the data from the Provincial Delivery Record. The data are then forwarded to the Northern and Central Alberta Outreach Program office located in Edmonton, Alberta.

Precautions are taken to ensure the accuracy of the data. The Audit Co-ordinator reviews records received in paper format for discrepancies before being entered into the database by a data entry clerk. A data validation process follows data entry and consists of a monthly crosscheck of the manual tabulation of key variables with an electronic tabulation of the same key variables. A minimum of 1 in 20 records is verified with the actual data entry to ensure accuracy. Methods for validating electronic data are also provided to participating hospitals. In addition, the Audit Co-ordinator of the NCAPAPD follows a validation process for electronically transferred data, which consists of electronic tabulation and comparison with the Monthly Statistical Report supplied with the data.

The data from the NCAPAPD used in this study included records from all women who delivered (a) liveborn infant (s) in participating hospitals in the Northern and Central Alberta Outreach Program during the study period, calendar years 1996, 1997, and 1998. The data fields as identified in the NCAPAPD database included: genetic and constitutional factors, pre-existing diseases, medical disorders, obstetrical history, problems in the current pregnancy, nutritional-related factors, pregnancy complications, lifestyle factors, maternal age, and selected information about the infant. Further detail are presented below.

There were both strengths and limitations associated with using this database. One strength was the large number of risk factors that could be examined, especially those related to maternal health. One of the limitations, however, was the way in which these variables were derived. With the exception of several obstetrical history variables (livebirths, previous PTB, abortions) and infant factors (gestational age, birth weight), the variables were aggregated and recorded as presence and absence of the risk factor. This means that precision was lost. An additional limitation was that the NCAPAPD did not include all births in Northern and Central Alberta. It captured only those births occurring in hospitals participating in the NCAPAPD during the study period. The number of hospitals participating in 1996, 1997, and 1998 were 35, 39, and 47 respectively. Ninety eight percent of births within the Northern and Central Alberta were captured and included in the database.

The Combined PNOB Data and Alberta Vital Statistics Data

The PNOB database contains a record for each birth in the Province of Alberta. It is derived from the provincial Notice of a Live Birth or a Still Birth and Newborn Record (PNOB) that is completed when an infant is born. The attending physician or midwife completes this record within 24 hours of birth. The Vital Statistics data are maintained by and reside in the Vital Statistics branch of Alberta Registries.

A copy of the Vital Statistics birth data is provided to the Health Surveillance Branch of Alberta Health and Wellness. Health Surveillance adds geocoding information and distributes the data within the health care sector. In the time period covered by this study, important data on the PNOB were not routinely entered into the electronic databases. In a project undertaken by the Health Surveillance Branch (Tough et al., 1999), these additional data were entered into an electronic database, and linked to the Vital Statistics records. The combined database, called the PNOB database, is maintained by Health Surveillance and was the source of the data for this study.

One strength of the PNOB database was the inclusion of geocoding fields that permit geographic area census data to be added. Another strength was that some lifestyle related factors (e.g., prenatal visits and prenatal class attendance) were only available in this database. A limitation of the database was that there were a substantial number of records for which data on street drug use, number of prenatal visits, and attendance at prenatal classes were missing.

Statistics Canada 1996 Census Public Use Microdata Files

The Statistics Canada 1996 Census Public Use Microdata Files were utilized to derive socio-economic indicators for specific geographic areas in CHA. The 1996 Census Data contains a large number of variables. However, the geographic resolution at which these data could be linked in the current study was low. In fact due to confidentiality of files only values at the PHSA level could be derived, rather than at the level of smaller neighbourhoods. Using Map Info Statistical PC Census Software, five specific census variables were used to represent the SES for each PHSA (percentage of individuals with less than grade nine education, percentage of persons with baccalaureate education or greater, median census family income, percentage of aboriginal population, and percentage of single parent families) (Capital Health Technical Report, 2000). These census variables were made available to the investigator in a hard copy. Appendix A provides the specific census data for each PHSA. The census variables chosen for the study were selected based on availability of the census data for CHA and the works of other researchers who investigated geographic variations in LBW (Crosse et al., 1997; O'Campo et al., 1997), indicators of social inequity (Pampalon & Raymond, 2000) and area-based socio-economic factors (Geronmius & Bound, 1998; Johnson, Drisko, Gallagher, & Barela, 1999; Parker et al., 1994).

Although numerous investigators are utilizing census-based socio-economic characteristics of residential areas to address the problem of inadequate individual socio-economic information on health data sets, there are limitations to their use (Diez-Roux, Nieto, Munatner, Tyroler, Cmstock, Shahar et al., 1997; Kreiger, 1992; O' Campo & Guyer, 1999; Rahkonen, Lahelma, & Huuhka, 1997, & Roberts, 1997). The first limitation of area-based measures is that area measures *assume* that the aggregate measures are indirect indicators of group properties that affect all persons within that group. The second limitation is that confounding by individual level variables or confounding at the contextual level may exist (Diez-Roux et al., 1997). For example, as in the former case, if women of lower education and lower income are at risk for LBW due to the neighbourhood they live in, then part of the neighbourhood effect may be adjusted away when control for the individual variables is taken into account. Confounding at the contextual level occurs as a result of individuals forming a part of a variety of contexts, many of which may overlap. For example, if the PHSAs are segregated based on people's relation to organization of work, persons within a neighbourhood may share a similar work environment, and characteristics of the environment may be related to LBW (Diez-Roux et al., 1997; Krieger, 1997).

A third limitation of using area-based SES measures is that measurement error can exist. Demissie, Hanley, Menzies, Joseph, and Ernst (2000) suggest that the misclassification associated with area-based measures is likely to be non-differential (i.e., the probabilities of exposure misclassification are the same in all groups being compared and unrelated to disease exposure). The consequence of such an unreliable estimate is to attenuate the exposure-outcome regression estimate, resulting in a failure to reach statistical significance.

The most important limitation of the SES data used in this study is that there were only a limited number of distinct data values for each measure (i.e., there are only 17 distinct values, one for each PHSA). This puts an absolute limit on the number of such variables that could be included in the analysis without introducing collinearity. This small number of distinct values also limits the extent to which the area-based measure can be expected to distinguish differences.

Data Linkage and Data Assembly

The NCAPAPD and the PNOB databases each contain one record for each birth event occurring within the specified geographic area within the specified time period. However, there is no single common key in these records that would easily allow the assembly of information from both sources into a single record about a single birth event. Therefore, a data linkage process was required. This procedure is discussed in some detail below as it consumed a substantial amount of time and resources.

The two databases had fields that putatively contained the same or similar information. Decisions about how to handle this type of information, including rules to handle disagreements between data sources were required.

Linking NCAPAPD and PNOB

To prepare for data linkage it was important to examine the databases for data that were both common and/or different. The data were examined to identify consistency of data definitions and coding from year to year (1996-1998). For ease of management of the data set, a special variable-naming convention was employed. For variables contained only in one database, the name was related to the variable content. For variables contained in both databases the same prefix (smk_) was used for those variables that measured roughly the same thing between the databases. The source of the data was identified by assigning a unique suffix to the NCAPAPD database (pn) and different ones to the combined Alberta Vital Statistics (vs) and PNOB data (ob). For instance, for the smoking history variable the prefix was smk_, and the variables became smk_pn, denoting NCAPAPD and smk_ob, denoting Alberta Vital Statistics/PNOB.

These databases were linked selecting identifiers common to the databases (Newcombe, 1988). In the simplest and least problematic cases, a unique identifier such as an ID number on each file allows straightforward linkage. When no ID number is available, the analyst must rely upon other information to link records together, and the possibility will always exist that an incorrect match will be made even with an exact match of the linking information. For example, if name information is being used to link records, it is possible that with frequent names like Mary Smith, the wrong records will be inadvertently connected. As well, there may be spelling and transcription errors that prevent an exact match in the linkage variables, even though it is highly likely that the two records are the same individual (e.g., Mary Jane McFinn and Mary Jane MacFinn).

The goal in linkage procedures is to make the maximum number of correct matches while minimizing incorrect matches. There are generally two ways to proceed. In deterministic linkage, a series of rules for what constitute a match are created, and matches by these rules are accepted as correct. The challenge is to create the appropriate rules and the appropriate sequence for the application of these rules to maximize correct matches and minimize incorrect matches. In probabilistic linkage, a probability of matching is calculated (based on a large collection of unmatched records and a large collection of known-to-be properly matched records) to help guide the application of the rules. A match according to a rule would be accepted if and only if the ratio of correct matches to incorrect matches was sufficiently high. In the current analysis, the deterministic linkage approach was utilized. The linkage between the databases was performed using custom-built Fox Pro software.

The first step in the linkage was to determine the data fields putatively containing the same information independently within each data file. In the current case the variables were: maternal age, gestational age, date and time of birth, infant weight, gender, and number of living children. To the extent that the combined set of information is complete and accurate in both data files and sufficient jointly to uniquely identify a single record, a set of rules can be generated to link records from the two sources together. To the extent that these conditions are not met, either there will be unlinked records, or errors in the data linkage will occur. In general, in a deterministic record linkage a succession of rules is generated by which records are provisionally matched. Typically, each successive rule relaxes some criterion for considering records to be matched. For example, if the last name was one of the fields for matching, a rule might be formulated which allowed minor spelling errors to occur at some stage in the linkage process.

There were nine rules developed for matching the records. These rules were applied separately in sequence, and records matched at each stage were considered to be properly linked. Table 1 identifies both the rules and the number of records that were matched.

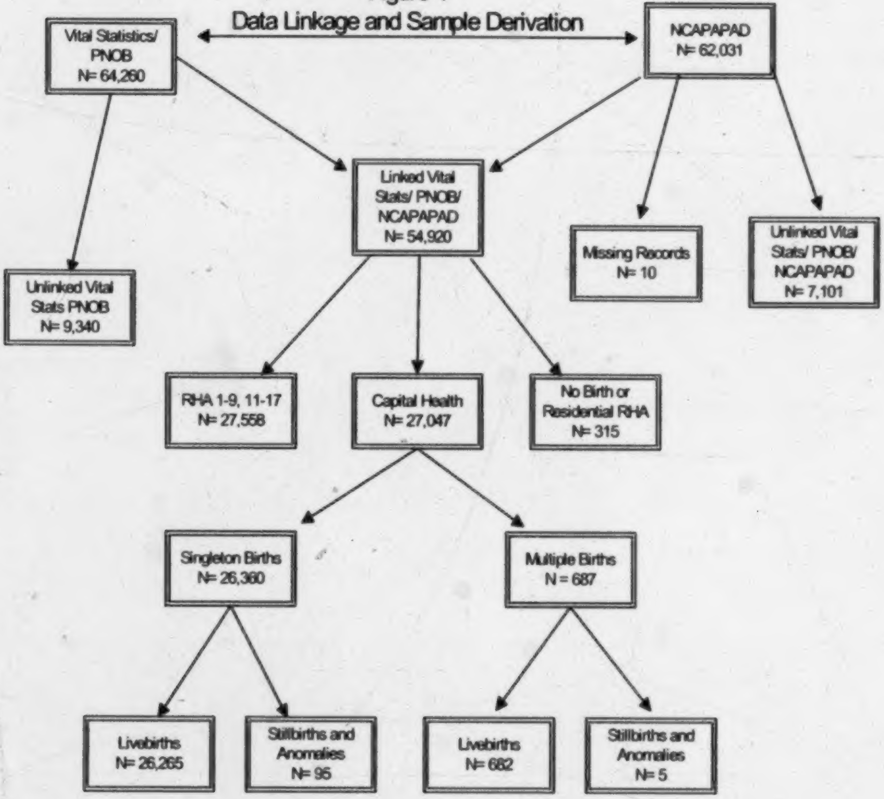
Table 1

Deterministic Linkage Between NCAPAPD and the Combined PNOB/Vital Statistics Data

Step	Linkage Rules							Matches
	Date of Birth	Gestational Age	Birth Weight	Sex	Maternal Age	No of Living		
1	√	√	√	√	√	√		41,818
2	√	√	√	√	√	Not matched		3,037
3	√	√	√	√	Within 1 year or missing	Not matched		2,632
4	√	Within 1 week	√	√	Within 1 year or missing	Not matched		5,498
5	√	√	Missing	√	√	Not matched		346
6	√	√	<10 g	√	√	Not matched		403
7	√	√	<10 g	Missing	√	√		293
8	Within 1 day	√	√	√	√	Not matched		245
9	√	Missing	√	√	Within 1 year or missing	Not matched		648
								54,920

The total number of NCAPAPD records was 62,031 and the total number of records included in the combined PNOB data and Alberta Vital Statistics Data was 64,260. The total number of linked records was 54,920--a linkage rate of 88.5%. Of the 54,920 records there were also 315 records eliminated from the analysis because they did not contain the birth Regional Health Authority and/or the residence Regional Health Authority. The birth and residential regional health authority were required to identify the women as residents of CHA and to complete the socio-economic analysis of the study.

Figure 4
Data Linkage and Sample Derivation


Vital Statistics/ PNOB N= 64,260
NCAPAPAD N= 62,031
Linked Vital Stats/ PNOB/ NCAPAPAD N= 54,920
Unlinked Vital Stats PNOB N= 9,340
Missing Records N= 10
Unlinked Vital Stats/ PNOB/ NCAPAPAD N= 7,101
RHA 1-9, 11-17 N= 27,558
Capital Health N= 27,047
No Birth or Residential RHA N= 315
Singleton Births N= 26,360
Multiple Births N = 687
Livebirths N= 26,265
Stillbirths and Anomalies N= 95
Livebirths N= 682
Stillbirths and Anomalies N= 5

This study included only liveborn singleton births with no anomalies who were resident within the CHA. Consequently all multiple births, stillbirths, and infants with major anomalies were excluded from the final data set. A total of 27,558 records from other RHAs were eliminated. Of the CHA records, 687 multiple births were eliminated (5 of which were stillbirths). In addition, 35 singleton stillbirths, 58 singleton liveborn infants with a major congenital anomaly, and 2 singleton stillbirths with a major congenital anomaly were also eliminated. The final data set for analysis contained 26,265 matched records. Figure 4 displays the final matched data set and identifies the distribution of singleton births, multiple births, stillbirths, and congenital anomalies.

Census Data

Census data were assigned to each birth record that had an identified PHSA. The census data did not require a linkage process as the data were provided to the investigator in paper format. Variables were created for each specific census variable. As a result, each woman within each PHSA was assigned the value of her respective PHSA for each census variable. For example, all women in Central PHSA were assigned a median census family income of $30,000, whereas all women in St. Albert were assigned a median census family income of $62,000. The variables used in the study included median census family income, percentage of aboriginal population, percentage of single parent population, percentage of population with baccalaureate degree or greater, and percentage of population with less than a grade nine education.

The Assembled Data File

The assembled file included data fields for linkage information (NCAPAPD & PNOB), genetic or constitutional factors (NCAPAPD), obstetrical history (NCAPAPD & PNOB), prenatal care factors (PNOB), lifestyle factors (NCAPAPD & PNOB), pre-existing medical diseases (NCAPAPD), nutritional problems during pregnancy (NCAPAPD), medical problems during pregnancy (NCAPAPD), pregnancy complications (NCAPAPD), indications for induction and operative delivery (NCAPAPD), additional information regarding infant characteristics (NCAPAPD & PNOB) and birth characteristics (NCAPAPD), maternal age (NCAPAPD & PNOB), geographic data for RHAs and PHSAs (PNOB), and socio-economic factors (CENSUS). Appendix B provides a table summary of the assembled file with a brief description of each variable and identification of the data source.

Data Transformations

Data transformations of the variables of the assembled file were necessary for several reasons: a) to address disagreements between variables available from multiple sources (infant birth weight, gestational age, maternal age, live births); b) to create new variables from existing variables in one or more of the data sets (i.e., smoking, alcohol, number of abortions, gestational hypertension with proteinuria, diabetes mellitus, heart disease, chronic hypertension); c) to develop cut points (i.e., stillbirths, abortions, maternal age, live births, infant birth weight, gestational age, median census family income, less than grade nine education, baccalaureate degree education or greater, single parent population, aboriginal population); d) to redefine geographical boundaries (i.e., residential RHA, birth RHA, PHSA, RHA boundary identification); e) to address missing data issues (prenatal visits, prenatal classes, use of street drugs); f) to rename data fields to be interpretable (i.e., d5=inappropriate or excessive drug use was renamed druguse); and g) to assign presence of the risk factor as 1 and absence of the risk factor as 0.

All variables resulting from a transformation were named according to a unique naming convention that captured the grouping and the variable content. Appendix C provides the names of all the transformed or combined variables, identifies the original variable from the data assembled file, identifies the data source, and provides a brief description of the transformation rules. Appendix C contains both a discussion and tables of the specific data transformations and data combinations. For example, one transformation was the identification for each individual RHA followed by identification of the birth records of mothers within the PHSA of CHA. With this transformation it was found that of the 26,265 CHA women who were resident and gave birth to a liveborn singleton, there were 1,387 women who could not be identified to a specific PHSA of CHA. Consequently these women were assigned to an undetermined PHSA called PHSA #18.

The sections on census data transformations and missing data transformations are also of particular interest as they contain methodological contributions. For example, to avoid losing all of the data from subjects with missing data, the technique proposed by Cohen & Cohen (1984), which involves creating a new variable that indicates for each individual whether data were missing was used to transform the variables prenatal classes, prenatal visits, and street drug use.

The appropriate use of census data required transformation and analyses. Initially, each woman was assigned her specific PHSA value for each census data variable. Because of the limited number of variables that could be employed in the analysis, a principal component analysis was undertaken to determine if a smaller number of components (linear combinations) could both capture the interrelationships among the five census variables and be used in the analyses to replace them. It was discovered that two components were sufficient to represent the relationships between the 5 census variables. The first component represented low socio-economic status. The second component was associated with higher educational status and a higher proportion of single parent families, and thus was more difficult to interpret. This component might be understood to represent the characteristic of resourcefulness or stability independent of income. The results of these analyses are provided in Appendix C, Table 16 and Table 17.

Data Analysis

Descriptive analyses were performed followed by multivariate analyses that described the dynamics among the factors under study. First, characteristics of the study population were determined. Next, the prevalence of maternal risk factors within CHA as a whole and across each of the PHSAs were ascertained. Then, a comparison of selected risk factors and outcomes between the study population and the unlinked records were examined. Lastly, mapping of the distribution of the risk factors and the proportion of PTB and LBW births across the PHSAs was completed. The methods for this mapping consisted of several steps: a) calculation of rates for each service area; b) calculation of the rate for the region; c) calculation of the standard error of each service area; d) calculation of the service area specific standard scores; and lastly, e) presentation of the standard scores as a map. The methodology used for the mapping is consistent with the methods used by Alberta Health and Wellness Health Surveillance reports (Ellehoj, E. personal communication, August, 2003). Appendix D provides an in-depth description of the calculation and interpretation of geographic rates.

Multivariate logistic regression (MLR) was used to identify those maternal and newborn factors that were significant in predicting LBW and PTB, while simultaneously controlling for the effects of the other study variables (potential confounders). Four models were developed. The four models were developed both to determine if the risk factor estimates remained the same when aggregate SES factors were added to each of the LBW and PTB models and to permit joint examination of the PTB and LBW analyses. As a result, two models used PTB as the dependent variable, and two models used LBW as the dependent variable. To determine if the individual risk factors remained predictive when aggregate SES factors were considered, one of the PTB and one of the LBW models added aggregate SES factors to the basic set of risk factors. The use of 4 models was necessary because the General and SES models included different samples. The General PTB and LBW models included the 1,387 women assigned to PHSA #18 as data were available regarding their individual risk factors. Including these women in the general models would result in more precise risk estimates for the study variables. However, SES data were not available for these women so they were not included in the SES models. An adjusted odds ratio and the 95% confidence limit were derived for each of the factors in each model. Following separate examination of the PTB and LBW analyses, joint examination of the PTB analysis and the LBW analysis was completed to identify both the direct and indirect influence of the risk factors on LBW.

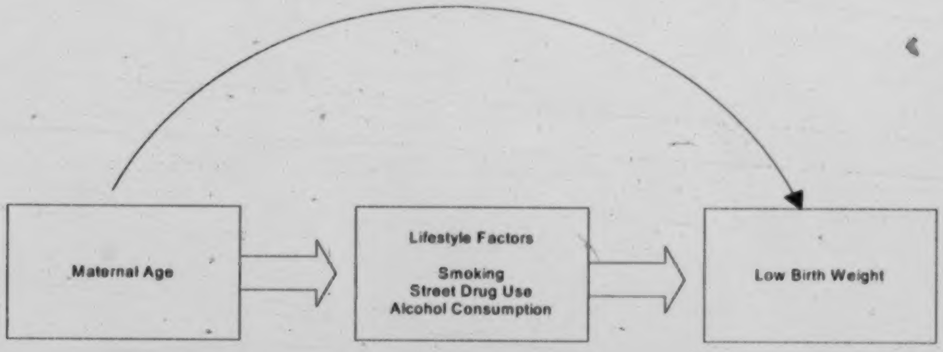
The general ordering of the risk factors in the MLR models was congruent with the hypothetical model developed from the literature review (see Figure 1). Socio-demographic, genetic and constitutional factors, and obstetric factors present prior to the index pregnancy (i.e., maternal age, pre-pregnancy height and weight, and obstetrical history) were sequentially entered. Next, prenatal care was entered, followed by those factors that could potentially be influenced by prenatal classes or prenatal visits (i.e., lifestyle, pre-existing medical diseases, nutritional factors, medical factors of pregnancy, and pregnancy complications).

Interaction terms were also considered for inclusion in the model. The selection of the interaction terms was based on previous research studies. The interaction terms that were examined included smoking by parity, smoking by age, alcohol by smoking, smoking by street drugs, parity by prenatal classes, and gestational hypertension by maternal age. Significant interactions existed for prenatal classes by parity, and maternal age by gestational hypertension. These interactions were entered in the model following the entry of the individual variables. For example, the prenatal class by parity interaction was entered separately following the entry of the obstetrical history and prenatal care variables.

Pregnancy complications have been identified as intermediate outcomes of PTB and LBW (Kramer, 1987; Lang et al., 1996), and, therefore they were entered last in the PTB model and prior to gestational age in the LBW model. By entering the pregnancy complications separately, it was possible to determine if the study risk factors influenced birth weight indirectly through these complications. Gestational age was the last variable to be entered in the LBW model. Gestational age was entered last to determine whether the effects of the risk factors on birth weight were direct or indirect (operating through the effects on gestational age).

Analysis is considerably simplified if a set of independent variables (block of variables) is conceptualized as ordered in a causal chain (Cohen & Cohen, 1984). This conceptualization permits examination of the influences of more distal variables on proximal variables.

Figure 5. Causal Chain of Risk Factors and LBW



As illustrated in Figure 5, the variable in the first block (maternal age) are understood to influence the dependent variable (LBW) or any block of variables along the chain (e.g., lifestyle factors). This diagram represents a causal chain of the influences on the dependent variable LBW. The over-arching arrow between maternal age and LBW represents the direct effect of maternal age on LBW. The arrows between maternal age and lifestyle, and between lifestyle and LBW, demonstrate the indirect effect of maternal age on LBW. That is, lifestyle factors mediate the relationship between maternal age and LBW. The blocks farther to the left are termed distal in part because they are farther away from the event in time; the factors closer in time and closer to the right are called proximal. Among the variables within a block, a causal ordering is not hypothesized either because the variables act simultaneously, the variables are involved in a complex interdependent cycle of causation, or insufficient information exists to speculate on the causal ordering.

There were 17 blocks of variables. Figure 6 depicits all the blocks and their constituent variables for the General LBW model. The General PTB model is identical to Figure 6, with the deletion of Block 17 (Gestational Age). For both the PTB and the

LBW Socio-economic models, the Socio-economic variables (Factor 1 and Factor 2) were inserted between Block 1 (Maternal Age) and Block 2 (Genetic and Constitutional Factors).

Figure 6. Order for Entry of Variables into the General Low Birth Weight Model

Block 1 Maternal Age (younger and older)

↓

Block 2 Genetic and Constitutional (pre-pregnancy height and weight)

↓

Block 3 Obstetrical History (parity, stillbirth, abortions, previous PTB, neonatal deaths, Small for Gestational Age infant)

↓

Block 4 Prenatal Care (prenatal visits and classes)

↓

Block 5 Prenatal Classes x Parity Interaction

↓

Block 6 Lifestyle Factors (smoking status, alcohol consumption and street drug use)

↓

Block 7 Alcohol x Smoking Interaction

↓

Block 8 Pre-existing Medical Diseases (heart disease, diabetes mellitus, renal disease and other medical diseases such as epilepsy, severe asthma, lupus, Crohn's disease)

↓

Block 9 Nutritional Problems During Pregnancy (poor gestational weight gain, anemia)

↓

Block 10 Medical Problems During Pregnancy (gestational diabetes, poly/oligohydramnios/ acute medical problems such as urinary tract infection, acute asthma, thyrotoxicosis)

↓

Block 11 Pregnancy Complication (gestational hypertension)

↓

Block 12 Gestational Hypertension x Maternal Age Interaction

↓

Block 13 Pregnancy Complication (gestational hypertension with proteinuria)

↓

Block 14 Pregnancy Complication (bleeding less than 20 weeks)

↓

Block 15 Pregnancy Complication (bleeding greater than or equal to 20 weeks)

↓

Block 16 Pregnancy Complication (placenta previa)

↓

Block 17 Gestational Age

↓

LBW

In the MLR analysis, variables were entered sequentially in blocks as indicated in Figure 6. In each model, the dependent variable (LBW or PTB) was regressed first on the variables in the most distal block (Block 1: maternal age). The next step involved the regression of the dependent variable on the variables in both Blocks 1 and 2. This procedure was followed in subsequent analyses until the variables from all blocks were simultaneously entered into a single analysis.

The advantage of this form of analysis is that comparison of any analysis with the next analysis will give an indication of the extent to which the more distal variables influence the more proximal variables, and the extent to which the more distal variables maintain a direct effect on the dependent variables. In the case where examination of the coefficients or odds ratios shows a change when the more proximal block is entered, this indicates that the effects of the more distal variables (as indicated in the previous analysis) are mediated by the variables in the more proximal block. That is, the effects of the more distal variables are indirect and operating through the proximal variables. Thus, indirect effects were determined when a change in the apparent effect size was noted when a new block of variables was entered.

Another way of expressing this situation is to suggest that the more distal variables are 'confounders' of the relationship between the proximal variables and the dependent variables; or, conversely, that the more proximal variables are confounders of the relationship between the distal variables and the dependent variable. Unfortunately, this form of analysis is partly qualitative in nature because precise numerical quantities cannot generally be given for 'blocks' of variables. That is, it is not possible to determine the effect of the total block of variables. In order to do this, a fully recursive model would be required; that is, every variable would need to be positioned uniquely within the chain. This would result in a path analysis (Cohen & Cohen, 1984).

Ethical Considerations

The security of the subject's file record was protected throughout the linkage process. The NCAPAPD database did not include personal identifiers. The original PNOB and Alberta Vital Statistics records were not available to the investigator. A representative of the Health Surveillance Branch of Alberta Health and Wellness completed all data linkages under the supervision of the author. The linked database that was provided to the investigator did not include any personal identifiers. The data were requested in a detailed research proposal and received full approval from The Northern and Central Alberta Perinatal Outreach Program Committee and the University of Alberta, Capital Health, and Caritas Health Group Health Research Ethics Board (B: Health Research).

Chapter 4 Results

The results of the study are presented in several sections. First, the characteristics of the mothers and infants and the maternal risk factor prevalences in CHA linked records are summarized. This is followed by a presentation of the results of the PTB and LBW models, highlighting differences between the general models for PTB and LBW and the models with the addition of socio-economic factors.

Characteristics of the Study Mothers and Infants

The study population consisted of 26,265 women who were resident in CHA and gave birth to a liveborn singleton between 1996 and 1998. Within the infant study population, 51.5% (n=13,522) were male, the mean birth weight was 3,384 grams (SD = 553 grams), and the mean gestational age was 38.9 weeks (SD=1.9 weeks). Overall, the total number of LBW infants was 1,269, representing 4.8% of all births within CHA. The percentage of preterm infants was 6.7 %. The mean maternal age was 28.3 years (SD = 5.5 years), 43% (n=11,373) of the mothers were nulliparous prior to this birth, and 57% (n=14,892) were multiparous.

Prevalence of Maternal Risk Factors in Capital Health

A key purpose of this study was to describe the prevalence of maternal risk factors both within CHA as a whole and within each of the PHSAs of CHA. Risk factor prevalences were restricted to maternal age, genetic and constitutional factors, obstetrical history, lifestyle characteristics, nutritional problems during pregnancy, prenatal care, pre-existing medical diseases, medical problems during pregnancy, and pregnancy complications. Table 2 presents the prevalence of these risk factors.

Adverse pregnancy outcomes such as prematurity and LBW have commonly been associated with younger pregnant women (women less than 20 years of age) and those of advanced childbearing age (35 years of age or older) (Jolly et al., 2000; Ziadeh, 2001). The maternal age of the study population ranged from 14 to 47 years with a mean of 28.3 years. Eighty percent of all births occurred among women aged 20-34 years. Among women younger than or equal to 19 years of age and women older than or equal to 35 years, the LBW rate was 7.0% and 5.3% respectively. A similar pattern was evident for

PTB; the younger maternal age group (younger than or equal to 19 years) and older maternal age group (35 years or older) had the highest percentages of PTB, 8.1% and 7.6% respectively.

The majority of women who delivered in the study period had no pre-existing medical diseases. Only 6.2% of the women entered their pregnancy with a history of one or more pre-existing medical diseases. Fewer than 2% of women had nutritional problems during their pregnancy (i.e., anemia or poor weight gain). Approximately 15% of the women had one or more pregnancy complications (i.e., placenta previa, gestational bleeding, gestational hypertension, pre-eclampsia, or premature rupture of membranes). About one third (36.5%) of the women in the study had a previous adverse pregnancy outcome (i.e., PTB, stillbirth, spontaneous or induced abortion, SGA birth, or neonatal death).

The overall prevalence of smoking in the study population was 26.2%. Smoking was more common among younger women, with nearly 61% of women under 20 reporting tobacco use, while fewer than 6% of women aged 35 and older reported usage. The prevalence of LBW among smokers was higher when compared with non-smokers, 7.6% versus 3.8% respectively. The combined effects of age and smoking on LBW were also noted. Women aged 12-19 years who smoked had a LBW rate of 8.2% compared to 10.6% of the women over 35 years who smoked. In terms of alcohol consumption and street drug use, 4.6% of mothers consumed alcohol and 2.1% of the women used street drugs during their pregnancy.

Prenatal care is generally conceptualized as including prenatal visits. For the purpose of this study, both prenatal visits and prenatal class attendance are included as components of prenatal care because both the visits and the classes provide potential opportunities for preventive counselling that may reduce risk factors for adverse birth outcomes. The database only provided the number of prenatal visits. It was not possible to identify the content or quality of prenatal care received by the mother during the prenatal visits. Nor was it possible to assess whether women actually participated in any preventive activities. The results of this analysis indicate that 70.3% of the women had eight or more prenatal visits.

The prenatal class participation rate within CHA was low. Only 30.2 % of the women in the study population attended prenatal classes. Differences in attendance were noted with respect to parity. Among multiparous women, 17.3% attended prenatal classes in comparison to 54.8 % of nulliparous women. Multiparous women may be less likely to attend prenatal classes because they have attended classes during a previous pregnancy. Prenatal class attendance also differed by maternal age, with the greatest attendance by women 20-34 years of age. Attendance rates for women younger than or equal to 19 years, 20-34 years, and 35 years or older were 6.2%, 82.6%, and 11.1% respectively.

Table 2

Prevalence of Maternal Risk Factors of Mothers Who Delivered a Singleton Liveborn Infant in Capital Health 1996-1998

Risk Factor	n	%	N
Demographic Characteristics			26,265
Maternal Age (years)			
≤ 19	1,697	6.5	
≥ 35	3,547	13.5	
Genetic or Constitutional Factors			26,265
Pre-pregnancy Weight			
≤45 kg	109	0.4	
≥ 91 kg	1,921	7.3	
Height			
< 152 cm	633	2.4	
Pre-existing Medical Diseases			26,265
Diabetes Mellitus	251	1.0	
Heart Disease	145	0.5	
Chronic Hypertension	242	0.9	
Renal Disease	27	0.1	
Other Medical Diseases (e.g., epilepsy, Crohn's disease, severe asthma, lupus)	963	3.7	
Obstetrical History			26,265
Previous Preterm Birth			
≥ 1	1,058	4.0	
Previous Abortions (spontaneous or induced)			
≥ 1	7,846	29.9	
Previous Neonatal Death	187	0.7	
Previous Stillbirth	356	1.3	
Parity			
0 (nulliparous)	11,373	43.3	
Small for Gestational Age Birth (SGA)	168	0.6	

Table 2 (cont'd)

Prevalence of Maternal Risk Factors of Mothers Who Delivered a Liveborn Singleton Infant in Capital Health 1996-1998

Risk Factor	n	%	Total N
Medical Problems During Current Pregnancy			26,265
Poly/Oligohydramnios	598	2.2	
Gestational Diabetes	902	3.4	
Acute Medical Problems (e.g., acute asthma, urinary tract infection, thyrotoxicosis)	101	0.4	
Nutritional Problems During Pregnancy			26,265
Poor Gestational Weight Gain (< 0.5 kg/wk weight gain or a weight loss between 26-36 weeks)	297	1.1	
Anemia (Hgb <100g/L)	91	0.3	
Pregnancy Complications			26,265
Placenta Previa	77	0.3	
Gestational Bleeding			
< 20 weeks	986	3.8	
≥ 20 weeks	732	2.8	
Gestational Hypertension	1,022	3.9	
Gestational Hypertension with Proteinuria (pre-eclampsia)	288	1.1	
Rupture of Membranes (spontaneous rupture of membranes before 37 weeks)	811	3.1	
Lifestyle Factors			26,265
Alcohol Consumption	1,200	4.6	
Use of Street Drugs	540	2.1	
Missing Use of Street Drug Data	1,005	3.7	
Smoking During Pregnancy	6,876	26.2	
Quit Smoking During Pregnancy	649	2.5	
Prenatal Care			26,265
Prenatal Classes			
No Prenatal Classes	14,661	55.8	
Missing Prenatal Classes Data	3,686	14.0	
Prenatal Visits			
≤ 4 visits	2,842	10.8	
5-7 visits	2,242	8.5	
Missing Prenatal Visits Data	2,730	10.4	

Comparison of Birth Outcomes and Risk Factor Prevalence in Linked and Unlinked Records

As noted in the description of the linkage process, there were 7,101 records that could not be linked. To allow for an assessment of whether a selection bias may have been introduced, the linked data were compared with the unlinked data set. To make appropriate comparisons between the linked and unlinked data, all multiple births, stillbirths, births with a major fetal anomaly, and missing data (i.e., missing gestational age, missing birth weight) were excluded from the unlinked records. Of the 7,101 unlinked records, there were 707 records that were excluded. A total of 6,394 unlinked records were included in this analysis.

Table 3 indicates that there were several significant differences between the women in the study sample and women in the unlinked records. Women in the unlinked data set were more likely to have pregnancy complications (i.e., gestational bleeding less than 20 weeks, gestational hypertension), a previous preterm birth, and to have smoked during their pregnancy. However, these women were less likely to consume alcohol and to use drugs. The mean gestational age of the infant was lower in the unlinked data; however, there were no significant differences in birth weight. These findings suggest that if the women in the unlinked group were residents of CHA, the prevalence of the selected risk factors within CHA would probably be higher. Thus, the reported odds ratios for several risk factors were likely underestimated.

Table 3
Comparison of Maternal Risk Factors and Infant Outcomes for Linked and Unlinked Records

	Linked (N=26,265)	Unlinked (N=6,394)	
Infant Outcome			
Gestational Age (mean weeks)	38.96	38.68	*
Birth Weight (mean grams)	3384.47	3417.36	NS
Maternal Risk Factors			
Maternal Age (mean years)	28.36	26.79	NS
Smoking (%)	26.2	29.5	**
Alcohol Consumption (%)	4.6	2.5	**
Use of Drugs (%)	2.1	0.8***	*
Gestational Bleeding (%)			
< 20 weeks	3.8	4.9	**
≥ 20 weeks	2.8	3.2	NS
Gestational Hypertension (%)	3.8	4.6	*
Previous Preterm Birth (%)	4.0	5.7	**

* Significant p<.05
** Significant p<.0001
NS Not Significant
*** Use of drugs in the unlinked data included prescription and non-prescription drugs. In the linked data use of drugs was specific to street drug use.

Comparisons were also made between the 1,387 women in CHA without geocoding for a PHSA (defined as PHSA #18) and the 6,394 women in the unlinked records without identifying RHA information. It was proposed that the absence of specific geographic information for both groups of women may be due to a woman's transience, poorer health, or environmental factors that may affect a woman's general health and lifestyle behaviors. It was thought that these two groups of women may be similar with respect to birth outcomes and risk factors. Table 4 provides a summary of the comparisons between linked PHSA #18 and unlinked records. The analysis revealed that while there were similarities, there were also significant differences between the women in PHSA #18 and the women in the unlinked data. The reason for these differences remains uncertain.

Table 4

Comparison of Maternal Risk Factors and Infant Outcomes for Linked Public Health Service Area # 18 and Unlinked Records

	Linked PHSA #18 (N=1,387)	Unlinked (N=6,394)	
Infant Outcomes			
Gestational Age (mean weeks)	38.64	38.68	*
Birth Weight (mean grams)	3290.43	3417.36	NS
Maternal Risk Factors			
Maternal Age (mean years)	26.74	26.79	**
Smoking (%)	41.7	29.5	**
Alcohol Consumption (%)	9.4	2.5	**
Use of Drugs (%)	7.5	0.8***	**
Gestational Bleeding (%)			
< 20 weeks	3.5	4.9	*
≥ 20 weeks	3.5	3.2	NS
Gestational Hypertension (%)	4.1	4.6	NS
Previous Preterm Birth (%)	5.2	5.7	NS

*Significant $p<.05$

**Significant $p<.0001$

NS Not Significant

*** Use of drugs in the unlinked data included prescription and non-prescription drugs. In the linked data use of drugs was specific to street drug use.

Results of Multivariate Analyses: Risk Models for Preterm Birth and Low Birth Weight

Multivariate logistic regression was used to develop four risk models: a PTB model with and without SES; and a LBW model with and without SES. In the following discussion, each model and its constituent risk factors are presented. The models are compared, and both the direct and indirect effects of risk factors are identified and discussed.

General Model: Preterm Birth

Table 5 details the final risk model for preterm birth with all variable blocks entered into the regression equation. The adjusted ORs and 95% CI for each study factor are provided in the table. Appendix E presents the results of the sequential analyses when each block of variables was entered into the model.

Table 5

Adjusted Odds Ratios for Maternal Risk Factors and Preterm Birth

Variable	OR	95% Confidence Interval
Maternal Age		
≤ 19 years	0.89	0.72 - 1.10
20-34 years	1.00	
≥ 35 years	1.16	0.99 - 1.36
Genetic or Constitutional Factors		
Pre-pregnancy Weight		
≤ 45 kg	0.80	0.35 - 1.87
46-90 kg	1.00	
≥ 91 kg	0.89	0.72 - 1.10
Height < 152 cm	1.05	0.76 - 1.40
Obstetrical History		
Previous Abortions (spontaneous or induced)		
≥ 1	1.11	0.99 - 1.24
Previous Preterm Birth		
≥ 1	4.33*	3.58 - 5.23
Parity		
Nulliparous	1.66*	1.24 - 2.22
Previous Stillbirth	0.85	0.58 - 1.23
Previous Neonatal Death	1.06	0.67 - 1.68
Previous Small for Gestational Age (SGA) Birth	1.37	0.81 - 2.31
Prenatal Care		
Prenatal Visits		
≤ 4 visits	4.61*	4.00 - 5.30
5-7 visits	4.05*	3.46 - 4.74
≥ 8 visits	1.00	
Missing Prenatal Visits Data (assumed to be .18)	0.97	0.83 - 1.14
Prenatal Classes		
Non-attendance	1.49*	1.13 - 1.96
Attendance at Prenatal Classes	1.00	
Missing Prenatal Classes Data (assumed to be .22)	1.09	0.92 - 1.28
Prenatal Classes x Parity Interaction:		
Nulliparity	1.06	0.77 - 1.46
Lifestyle Factors		
Smoking During Pregnancy	0.97	0.85 – 1.10
Quit Smoking During Pregnancy	0.96	0.68 – 1.36
No Smoking	1.00	
Alcohol Consumption During Pregnancy	0.61	0.36 – 1.04

*Significant p <.05

Table 5 (cont'd)
Adjusted Odds Ratios for Maternal Risk Factors and Preterm Birth

Variable	OR	95% Confidence Interval
Lifestyle Factors		
Use of Street Drugs	1.36*	1.00 – 1.84
Missing Use of Street Drug Data (assumed to be .33)	1.08	0.74 - 1.57
No Use of Street Drugs	1.00	
Alcohol x Smoking Interaction	2.14*	1.18-3.85
Pre-existing Medical Diseases		
Heart Disease	1.56	0.86 - 2.83
Diabetes Mellitus	2.13*	1.42 - 3.19
Chronic Hypertension	1.68*	1.12 - 2.53
Renal Disease	4.14*	1.62 - 10.54
Other Medical Diseases (e.g., epilepsy, severe asthma, lupus, Crohn's disease)	1.26	0.98 - 1.61
Nutritional Problems During Pregnancy		
Poor Gestational Weight Gain (< 0.5kg/wk weight gain or weight loss between 26-36 weeks)	1.03	0.65 - 1.63
Anemia (Hgb <100 g/L)	1.17	0.57 - 2.40
Medical Problems During Current Pregnancy		
Acute Medical Problems (e.g., acute asthma, UTI, thyrotoxicosis)	2.98*	1.70 - 5.19
Poly/Oligohydramnios	4.03*	3.21 - 5.06
Gestational Diabetes	1.29	0.99 - 1.67
Pregnancy Complications		
Gestational Hypertension	1.86*	1.40 - 2.46
Gestational Hypertension x Maternal Age Interaction		
≤ 19 years	0.35*	0.14 - 0.87
20-34 years	1.00	
≥ 35 years	1.68	0.99 - 2.84
Gestational Hypertension with Proteinuria (pre-eclampsia)	3.60*	2.45 - 5.30
Gestational Bleeding		
< 20 weeks	1.75*	1.40 - 2.18
≥ 20weeks	5.05*	4.15 - 6.13
Placenta Previa	6.97*	3.98 - 12.19

*Significant p <.05

The adjusted ORs and 95% CI for the study risk factors are detailed in Table 5. PTB was associated with obstetrical factors (previous PTB and nulliparity), pre-existing medical diseases (diabetes mellitus, chronic hypertension, and renal disease), medical problems during the current pregnancy (polyhydramnios/oligohydramnios, acute medical problems), and pregnancy complications (gestational hypertension, gestational bleeding, and placenta previa). As well, PTB was associated with several modifiable risk factors (e.g., lack of attendance at prenatal classes, reduced number of prenatal visits, use of alcohol in combination with smoking, and use of street drugs).

The ORs for pre-existing medical diseases ranged from 1.68 (CI: 1.12-2.53) for chronic hypertension to 4.14 for renal disease (CI: 1.62-10.54). Odds ratios for other medical problems during pregnancy ranged from 2.98 (CI: 1.70-5.19) for acute medical problems to 4.03 (CI: 3.21-5.06) for polyhydramnios/oligohydramnios. It was not possible to determine separate risk estimates for the different medical conditions (i.e., acute asthma, thyrotoxicosis, and urinary tract infection) because of the aggregation of these variables into one category.

All the pregnancy complications were significant predictors of PTB, with ORs ranging from 1.75 (CI: 1.40-2.18) for gestational bleeding less than 20 weeks to 6.97 (CI: 3.98-12.19) for placenta previa. Overall, gestational hypertension had a significant main effect (OR=1.86; CI: 1.40-2.46). However, there was also a significant maternal age by gestational hypertension interaction such that the effect of gestational hypertension was substantially attenuated in younger women (OR=0.35; CI: 0.14-0.87).

Additional maternal predictors of PTB included use of street drugs (OR=1.36; CI: 1.00-1.84) and prenatal care. Women who had fewer visits (≤ 4 visits or 5 to 7 visits) had a 4-fold risk of having a PTB. Women who did not attend prenatal classes had a 49 % greater odds of delivering a preterm infant (OR=1.49; CI: 1.13-1.96). Women who smoked and consumed alcohol during their pregnancy had a 2-fold risk of delivering a preterm infant (OR=2.14; CI: 1.18-3.85).

Smoking alone did not have a direct effect on PTB. To understand this null finding, the sequential order of the blocks of variables before and after the lifestyle block were reviewed. The prenatal care block was entered before the lifestyle block of variables. This order was chosen because it was thought that prenatal care might result in a change in the lifestyle behaviors (e.g., quitting smoking). However, it may also be true that smokers had fewer visits or did not attend prenatal classes. Thus, it was thought that if the prenatal care block (i.e., prenatal visits and prenatal classes) was removed from the analysis, the effect of smoking on PTB might become more visible. Consequently, an additional analysis was conducted, in which the prenatal care block of variables was left out. In this sub-analysis, smoking had a small statistically significant increased risk (OR=1.18; CI: 1.05-1.28) for PTB when the smoking block was first entered. Smoking became non-significant (OR=1.12; CI: 0.99-1.27) with the addition of the complication bleeding greater than 20 weeks. This change in the risk estimate suggests that smoking has an indirect effect on PTB by increasing the woman's risk of gestational bleeding, which may result in medical intervention to deliver an infant before term.

Another important null finding was maternal age. Although maternal age did not have a direct effect on PTB, had an indirect effect. For example, we can see from comparing column 1 of Appendix E to column 4 of Appendix E that with the addition of the prenatal care block to the regression model, that the OR for younger maternal age was no longer significant. This change in the OR suggests that the effect of young maternal age is through inadequate prenatal care. For older women, the influence of age operated through the pregnancy complications. As each pregnancy complication was accounted for in the model, the risk estimate for maternal age decreased slightly. Appendix E column 1 shows that the OR for older maternal age was initially significant (OR=1.19, CI: 1.04-1.37), but the OR became non-significant with the addition of placenta previa (see Appendix E column 16).

Preterm Birth Model With Addition of Socio-Economic Factors

Appendix F provides the odds ratios and 95% confidence intervals for the sequential entry of blocks of variables in the PTB SES analysis. In comparing the ORs in column 16 of Appendix E to column 17 of Appendix F, it can be seen that the magnitude and direction of the risk estimates for the study variables remained quite similar with the addition of the socio-economic factors to the PTB model. Neither of the socio-economic risk factors (low SES (factor 1), resourcefulness and stability (factor 2)) had a direct effect on PTB. Examination of the slight shift in the OR for factor 1 (low SES) in column 4 compared to column 5 of Appendix F suggests that the effect of low SES on PTB may be indirect, operating through its direct effect on prenatal care.

There were three factors that were not significant in the General PTB model that were significant independent predictors in the PTB SES model. Other medical diseases (OR=1.29; CI: 1.00-1.70) and gestational diabetes (OR=1.34; CI: 1.02-1.72) each had a direct effect in the PTB SES model. In addition, older women with gestational hypertension had a statistically significant increased risk of PTB (OR=1.78; CI: 1.03-3.06). Gestational hypertension had a significant main effect (OR=1.82; CI: 1.35-2.40). Although the effect of gestational hypertension was substantially attenuated in younger women (OR=0.54; CI: 0.21-1.40), this finding was not significant.

Table 6 identifies those risk factors which had a direct (independent) and/or indirect effect (operating through another risk factor) on PTB. Maternal age exerted indirect effects in both the General PTB and PTB SES models. Examination of column 3 and 4 in Appendix E reveals that in the General PTB model, young maternal age operates through prenatal care and older maternal age operates through complications of pregnancy. Examination of the maternal age coefficient in columns 2 and 5 in Appendix F suggest that in the PTB SES model young maternal age operates through both SES and prenatal care. Consistent with the General PTB model, the *most* substantial decrease in the maternal age risk estimates in the PTB SES model was when the prenatal care block of variables was added to the logistic regression equation. Similar to the General PTB model, the effect of older maternal age in the PTB SES model was through the occurrence of a pregnancy complication, specifically, gestational bleeding greater than or equal to 20 weeks.

Other medical diseases and gestational diabetes were only significant in the SES PTB model. However, in reporting the differences between the PTB models it should be noted that the change in the magnitude of the ORs for other medical diseases and gestational diabetes in the SES model was *very* small. Moreover, other medical diseases were *only* significant in the SES PTB model in the last step of the analysis, when all 17 blocks of variables were included. The different SES PTB model findings suggest that the effect of SES on PTB is indirect, operating through prenatal care, gestational diabetes, other medical diseases, and gestational hypertension by age interaction.

Table 6

Direct and Indirect Effects of Maternal Risk Factors for Preterm Birth Models

Risk Factor	General Preterm Birth Model		SES Preterm Birth Model	
	Direct Effect	Indirect Effect	Direct Effect	Indirect Effect
Maternal Age		√		√
SES	N/A	N/A		√
Obstetrical History				
Previous Preterm Birth	√*		√*	
Nulliparity	√*		√*	
Prenatal Care				
Prenatal Visits				
≤ 4 prenatal visits	√*		√*	
5-7 prenatal visits	√*		√*	
Prenatal Classes				
no prenatal classes	√*		√*	
Lifestyle Factors				
Street Drug Use	√*			
Alcohol x Smoking Interaction	√*		√*	
Pre-existing Diseases				
Diabetes Mellitus	√*		√*	
Chronic Hypertension	√*		√*	
Renal Disease	√*		√*	
Other Medical Diseases			√*	
Medical Problems During Current Pregnancy				
Acute Medical Problems	√*		√*	
Poly/Oligohydramnios	√*		√*	
Gestational Diabetes			√*	
Pregnancy Complications				
Gestational Hypertension	√*		√*	

* Significant $p < .05$

Table 6 (cont'd)
Direct and Indirect Effects of Maternal Risk Factors for Preterm Birth Models

Risk Factor	General Preterm Birth Model Direct Effect	General Preterm Birth Model Indirect Effect	SES Preterm Birth Model Direct Effect	SES Preterm Birth Model Indirect Effect
Pregnancy Complications				
Gestational Hypertension x Age Interaction				
younger	(protective)*			
older age			√*	
Gestational Hypertension with proteinuria (pre-eclampsia	√*		√*	
Bleeding < 20 weeks	√*		√*	
Bleeding ≥ 20 weeks	√*		√*	
Placenta Previa	√*		√*	

*Significant p< .05

General Model: Low Birth Weight

Multivariate logistic models of LBW have not typically included gestational age because birth weight is known to be directly proportional to gestational age and thus gestational age is the largest single determinant of birth weight. That is, the earlier an infant is born, the larger the deficits in birth weight are likely to be. Researchers who do not include gestational age as a predictor of LBW in their models are unable to examine concurrently direct and indirect effects of risk factors on LBW. That is, it becomes much more difficult to appropriately recognize whether the effects of other factors operate directly on birth weight or indirectly on birth weight by lowering gestational age (i.e., early or preterm delivery). Gestational age was included in the LBW model in order to provide some insight as to the direct and indirect influence of risk factors on birth weight. Table 7 represents the final regression model. The adjusted ORs and 95% CI for the study risk factors are detailed in Table 7. This model indicates that gestational age is responsible for a significant portion of LBW (OR=31.28; CI: 26.91-36.36). Appendix G presents the sequence of analyses by which blocks of variables were entered into the model.

Table 7
Adjusted Odds Ratios for Maternal Risk Factors and Low Birth Weight

Variable	OR	95% Confidence Interval
Maternal Age		
≤ 19 years	0.88	0.68 - 1.16
20-34 years	1.00	
≥ 35 years	1.13	0.91 - 1.41
Genetic or Constitutional Factors		
Pre-pregnancy Weight		
≤ 45 kg	1.93	0.88 - 4.23
46-90 kg	1.00	
≥ 91 kg	0.42*	0.30 - 0.59
Height < 152 cm	1.47	0.99 - 2.19
Obstetrical History		
Previous Abortions (spontaneous or induced)		
≥ 1	1.03	0.89 - 1.21
Previous Preterm Birth		
≥ 1	2.31*	1.77 - 3.01
Parity		
Nulliparous	1.47	0.99 - 2.78
Previous Stillbirth	1.10	0.66 - 1.83
Previous Neonatal Death	1.15	0.61 - 2.15
Previous SGA Birth	8.73*	5.49 - 13.90
Prenatal Care		
Prenatal Visits		
≤4 visits	2.07*	1.71 - 2.51
5-7 visits	1.74*	1.40 - 2.17
≥ 8 visits	1.00	
Missing Prenatal Visits Data (assumed to be .18)	0.87	0.69 - 1.09
Prenatal Classes		
Non-attendance	1.09	0.75 - 1.59
Attendance at Prenatal Classes	1.00	
Missing Prenatal Classes Data (assumed to be .22)	1.03	0.83 - 1.28
Prenatal Classes x Parity Interaction		
Nulliparous	1.53*	1.00 - 2.33
Lifestyle Factors		
Smoking	2.02*	1.73 - 2.39
Quit Smoking During Pregnancy	0.93	0.59 - 1.45
No Smoking	1.00	
Alcohol Consumption	0.62	0.29 - 1.35

*Significant p <.05

Table 7 (cont'd)
Adjusted Odds Ratios for Maternal Risk Factors and Low Birth Weight

Variable	OR	95% Confidence Interval
Lifestyle Factors		
Use of Street Drugs	1.41	0.96 - 2.06
Missing Use of Street Drug Data (assumed to be .33)	0.87	0.54 - 1.43
No Use of Street Drugs	1.00	
Alcohol x Smoking Interaction	1.60	0.69 - 3.69
Pre-existing Medical Diseases		
Heart Disease	1.36	0.58 - 3.17
Diabetes Mellitus	0.61	0.32 - 1.19
Chronic Hypertension	2.11*	1.23 - 3.62
Renal Disease	2.03	0.55 - 7.48
Other Medical Diseases (e.g., epilepsy, severe asthma, Crohn's disease, lupus)	0.99	0.07 - 1.40
Nutritional Problems During Pregnancy		
Poor Gestational Weight Gain (< 0.5 kg/wk weight gain or weight loss between 26-36 weeks)	2.43*	1.50 - 3.91
Anemia (Hgb <100g/L)	2.40*	1.10 - 5.24
Medical Problems During Current Pregnancy		
Acute Medical Problems (e.g., acute asthma, UTI, thyrotoxicosis)	1.42	0.63 - 3.19
Poly/Oligohydramnios	5.78*	4.40 - 7.59
Gestational Diabetes	0.97	0.66 - 1.42
Pregnancy Complications		
Gestational Hypertension	2.05*	1.41 - 2.97
Gestational Hypertension x Maternal Age Interaction		
≤ 19 years	0.16*	0.03 - 0.68
20-34 years	1.00	
≥ 35 years	1.40	0.69 - 2.80
Gestational Hypertension with Proteinuria (pre-eclampsia)	2.20*	1.32 - 3.68
Gestational Bleeding		
< 20 weeks	1.49*	1.11 - 2.01
≥ 20 weeks	2.12*	1.62 - 2.79
Placenta Previa	0.90	0.47 - 1.73
Gestational Age		
Preterm (< 37 weeks)	31.28*	26.91 - 36.36
Term (≥ 37 weeks)	1.00	

*Significant p <.05

Several risk factors that were predictive of PTB were also predictive of LBW. For the LBW model, the risk estimates for these common variables were as follows: previous PTB (OR=2.31; CI: 1.77-3.01), four or fewer prenatal visits (OR=2.07; CI: 1.70- 2.51), five to seven prenatal visits (OR= 1.74; CI: 1.40-2.17), chronic hypertension (OR = 2.11; CI: 1.23-3.62), polyhydramnios/oligohydramnios (OR= 5.78; CI = 4.40-7.59), bleeding less than 20 weeks (OR=1.49; CI: 1.11-2.01), bleeding greater than or equal to 20 weeks (OR=2.12; CI =1.62-2.79), gestational hypertension (OR= 2.05; CI: 1.41-2.97), gestational hypertension by young maternal age (OR=0.16; CI: 0.03-0.68), and gestational hypertension with proteinuria (pre-eclampsia) (OR =2.20; CI: 1.32-3.68). In both the PTB model and LBW model, gestational hypertension had a significant main effect, and there was a significant maternal age by gestational hypertension interaction such that the effect of gestational hypertension was substantially attenuated in younger women. When comparing the magnitude of risk estimates in the LBW and PTB model, the increased magnitude of the risk estimates in the PTB model for previous preterm birth, prenatal visits, gestational hypertension with proteinuria (pre-eclampsia), and bleeding greater than or equal to 20 weeks demonstrate their significant effect on gestational age. For example, women with bleeding greater than or equal to 20 weeks had a 5-fold risk of PTB, whereas in the LBW model this risk was 2-fold. Women with inadequate prenatal visits had a 4-fold risk of PTB compared to the 2-fold risk in the LBW model.

There were five risk factors (previous SGA, smoking, poor gestational weight gain, anemia, the interaction of nulliparity and non-attendance at prenatal classes) that were unique predictors of LBW. Previous SGA birth (OR=8.73; CI: 5.49-13.90), smoking (OR=2.02; CI: 1.72- 2.38), poor gestational weight gain during pregnancy (OR= 2.43; CI = 1.50-3.91), and anemia during pregnancy (OR= 2.40; CI: 1.10-5.24) all had a direct effect on LBW. Although anemia was a significant predictor of LBW, this finding must be interpreted with caution given that it was significant only with the addition of the gestational hypertension by maternal age interaction and pre-eclampsia and then not again until the addition of gestational age. Nulliparous woman who did not attend prenatal classes were at slightly greater risk for delivering a LBW infant (OR=1.53; CI: 1.00-2.33). The dramatic effect of gestational age on birth weight is seen in the 31-fold increased risk of having a LBW if a woman delivers a preterm infant.

To determine the indirect impact of the risk factors on LBW, the odds ratios in the General PTB analysis (Appendix E) and the General LBW analysis (Appendix G) need to be examined jointly. First, the final column of the PTB analysis is examined; then, the final column in the LBW analysis is examined. Lastly, the final column (column 17) in the LBW analysis and the next to final column (column 16) in the LBW analysis are examined to determine if there is a shift in the odds ratios.

The indirect effect of the risk factors is best illustrated in the examination of the pregnancy complications. When examining the final column in the PTB analysis (column 16) and LBW analysis (column 17) together, we can see that the ORs for bleeding greater than 20 weeks in the preterm analysis was 5.05 (CI: 4.15-6.13) and in the LBW analysis was 2.12 (CI: 1.62-2.79). However, in the LBW analysis, prior to adjustment for gestational age (column 16), the OR for bleeding greater than or equal to 20 weeks was 4.65 (CI: 3.73-5.80). This shift in the OR from 4.65 to 2.12 for bleeding greater than or equal to 20 weeks illustrates the indirect effect of gestational bleeding on gestational age and consequently LBW. This pattern of change in the ORs as gestational age is added to the LBW model was also observed for other complications of pregnancy (gestational hypertension, bleeding less than 20 weeks, gestational hypertension with proteinuria (pre-eclampsia), placenta previa), poly/oligohydramnios, nulliparity, previous PTB, prenatal visits, acute medical problems, renal disease, smoking and alcohol consumption, and street drug use. It is important to remember that it is also possible that pregnancy complications and medical problems did not result in spontaneous preterm labor, but rather that these conditions may have led to medical intervention resulting in a PTB.

Another indirect effect noted was that of maternal age. In the LBW analysis we can see from Appendix G that, although younger maternal age was initially significant in the model, when the prenatal care variables (prenatal visits and prenatal classes) were added, younger maternal age was no longer associated with LBW. As in the PTB model, women of advancing maternal age were at increased risk for gestational bleeding.

LBW Model With Addition of Socio-Economic Factors

Direct and indirect effects on LBW were also determined in the SES LBW model by jointly examining the SES PTB model and the SES LBW model. This examination was completed following the same steps as in the general LBW model. Appendix H provides the odds ratios and 95% confidence intervals for the sequential entry of each block of variables in the SES LBW analysis.

As in the PTB SES model, when the socio-economic risk factors are first entered in the logistic equation, factor 1 (low SES) was initially significant (OR=1.18, CI: 1.11-1.25). Also consistent with the SES PTB model, with the addition of the prenatal care block of variables (column 5 of Appendix H), factor 1 (low SES) no longer appears to have a direct effect on LBW.

There were several differences noted between the two LBW models. In the SES model, women with a pre-pregnancy weight less than or equal to 45 kg were at increased risk (OR=2.51; CI: 1.15-5.44) of delivering a LBW infant, as were women who used street drugs (OR=1.71; CI: 1.13-2.59). In reporting the OR for low pre-pregnancy weight, it is also important to note the change in the statistical significance for this factor. In the SES model, low pre-pregnancy weight was significant until the Nutritional Block of variables (anemia & poor gestational weight gain) was added to the analysis. However, when gestational age was added to the analysis, the OR changed from a non-significant increased risk (OR=1.98; CI: 0.97–4.10) to a statistically significant increased risk (OR=2.51; CI: 1.15-5.44). This finding suggests an indirect effect of SES on LBW operating through low pre-pregnancy weight. Furthermore, it suggests a possible relationship between low-pre-pregnancy weight (an anthropometric proxy measure of nutritional status) and ongoing nutritional status during pregnancy (anemia, poor gestational weight gain). The consistent significant and increased OR estimate for street drug use in the SES LBW model suggests an indirect effect of SES on LBW operating through street drug use. With the exception of low pre-pregnancy weight and street drug use, those risk factors that had a significant independent effect (direct effect) in the SES model were also evident in the General LBW model. Furthermore, the OR estimates were comparable in magnitude and direction.

As in the general LBW analysis, maternal age did not have a significant direct effect on LBW. However, in the SES LBW analysis we can see from Appendix H that the effect of age for older women once again operated through pregnancy complications.

Table 8 summarizes the direct and indirect effects of risk factors for both the General LBW model and the SES LBW model. With the exception of pre-pregnancy weight less than or equal to 45 kg and use of street drugs, the direct and indirect effects were consistent between the two models. While the change in the OR for the combined effect of alcohol and smoking in the SES LBW suggests an indirect effect, it must be interpreted with caution, as this risk factor was not consistently statistically significant with the addition of each block of variables.

When comparing the models, it is important to acknowledge that the SES LBW and PTB models excluded the 1,387 women in CHA who did not have SES data (i.e., PHSA #18), whereas the General PTB and LBW models did include these women. Differences in the samples of the General and SES models could in part account for the differences between the SES and non-SES analyses.

Table 8

Joint Examination of the Direct Effects and Indirect Effects of Risk Factors on Low Birth Weight

Risk Factor	General LBW Model Direct Effect	General LBW Model Indirect Effect	SES LBW Model Direct Effect	SES LBW Model Indirect Effect
Maternal Age		√		√
SES				√
Genetic & Constitutional				
Weight ≤ 45 kg			√*	
Weight ≥ 91 kg	(protective)*		(protective)*	
Obstetrical History				
Previous Preterm Birth	√*	√	√*	√
Nulliparity		√		√
Previous SGA Birth	√*		√*	
Prenatal Care				
Prenatal Visits				
≤ 4 prenatal visits	√*	√	√*	√
5-7 prenatal visits	√*	√	√*	√
Prenatal Classes				
no prenatal classes x nulliparity	√*		√*	
Lifestyle Factors				
Smoking	√*		√*	
Use of Street Drug		√	√*	
Alcohol x Smoking Interaction		√		√
Pre-existing Diseases				
Chronic Hypertension	√*		√*	
Renal Disease		√		√
Nutritional Problems				
Anemia	√*		√*	
Poor Gestational Weight Gain	√*		√*	
Medical Problems During Current Pregnancy		√		√
Acute Medical Problems	√*	√	√*	√
Poly/Oligohydramnios				

*Significant p <.05

Table 8
Joint Examination of the Direct Effects and Indirect Effects of Risk Factors on Low Birth Weight

Risk Factor	General LBW Model		SES LBW Model	
	Direct Effect	Indirect Effect	Direct Effect	Indirect Effect
Pregnancy Complications				
Gestational Hypertension	√*	√	√*	√
Gestational Hypertension x Age Interaction				
younger age	protective*		protective*	
older age		√		√
Gestational Hypertension with Proteinuria (pre-eclampsia)	√*	√	√*	√
Bleeding < 20 weeks	√*	√	√*	√
Bleeding ≥ 20 weeks	√*	√	√*	√
Placenta Previa		√		√
Gestational Age	√*		√*	

*Significant p <.05

Summary of Major Findings

Predictors that were common across all four models were previous PTB, reduced number of prenatal visits (≤ 7 visits), chronic hypertension, poly/oligohydramnios, and pregnancy complications, specifically, bleeding less than 20 weeks, bleeding greater than 20 weeks, gestational hypertension, and gestational hypertension with proteinuria (pre-eclampsia). Also constant across the four models was the indirect effect of maternal age. In both PTB models, the effect of maternal age was indirect but operated differently. In the General PTB model, young maternal age exerted its effect through prenatal care, whereas in the PTB SES model, young maternal age appeared to operate through both SES and prenatal care. The indirect effects of maternal age in the SES LBW model and the General LBW model were consistent with the effects in the General and SES PTB models. In these models, older maternal age operated through pregnancy complications. In the General LBW model young maternal age operated through prenatal care; in the SES LBW model, young maternal age operated through SES and prenatal care.

Although there were risk factors that were common to all 4 models there were also risk factors that were unique to each specific model. Risk factors that were unique in the PTB models included pre-existing medical diseases (diabetes mellitus, renal disease), acute medical problems during pregnancy, nulliparity, placenta previa, as well as the lack of prenatal classes and alcohol consumption in conjunction with smoking. In the SES PTB model, gestational diabetes, gestational hypertension for older women, and other pre-existing medical diseases were unique predictors, however, street drug use was the only unique predictor in the General PTB model. In the PTB SES model, SES had an indirect effect on PTB, operating through prenatal care, gestational diabetes, gestational hypertension for older women, and other pre-existing medical diseases.

There were also predictors that were unique to the LBW models and unique to the SES LBW model. Unique predictors for the LBW models were previous SGA birth, poor gestational weight gain, anemia, smoking, and the nulliparity by prenatal class interaction. Low pre-pregnancy weight and street drug use were unique predictors in the SES LBW model. In the SES LBW model, SES exerted an indirect effect through prenatal care, low-pre-pregnancy weight, and use of street drugs. When comparing the LBW models with and without SES, factors that exerted an indirect effect were similar, with the exception of use of street drugs. These factors influenced LBW through gestational age.

Chapter 5 Mapping Geographic Variations

Displaying and Understanding Geographic Variations in Maternal Risk Factors and Infant Outcomes

One of the purposes of this study was to provide information to practitioners and policy makers about both the distribution of the risk factors and the birth outcomes within the seventeen PHSAs of CHA. Of the 26,265 women who gave birth during the study period, there were 1387 women (5.2%) who were residents of CHA who could not be assigned to one of the 17 PHSAs. Although the women were missing PHSA socio-economic data, the remaining risk factor data were available. Consequently, these women were included in the study and assigned to a fictitious PHSA called PHSA #18.

Examination of the data for risk factors and birth outcomes for each PHSA revealed considerable geographic variations. Table 9 shows that the distribution of LBW and PTB infants differs across the PHSAs. Only Central PHSA and PHSA #18 had a LBW prevalence above the national LBW rate of 5.7% (6.9% and 7.6% respectively). However, it is important to note that the national LBW rate includes multiple births, stillbirths, and infants with congenital anomalies and therefore is not directly comparable.

Table 9

Capital Health Public Health Service Area Low Birth Weight and Preterm Birth Rates

Public Health Service Areas Capital Health Region	Low Birth Weight Rate % (N)	Preterm Birth Rate % (N)
St Albert	3.5 (50)	4.5 (64)
Castle Downs	3.6 (47)	5.7 (75)
West Central	4.7 (88)	6.9 (129)
Central	6.9 (124)	8.6 (155)
North Central	5.4 (101)	6.8 (127)
North East	5.2 (126)	6.2 (150)
West (North)	5.6 (120)	7.3 (157)
West (South)	3.8 (46)	5.6 (67)
South West (West)	4.1 (57)	6.3 (88)
South West (East)	4.2 (62)	6.5 (96)
South Central	3.5 (57)	6.2 (103)
Millwoods	5.3 (185)	6.6 (231)
Sherwood Park	3.4 (44)	7.1 (92)
Strathcona County	3.6 (19)	4.7 (25)
Thorsby	2.6 (6)	6.1 (14)
Leduc	4.0 (19)	6.4 (30)
Beaumont	4.6 (12)	5.8 (15)
PHSA #18	7.6 (106)	9.9 (137)
Capital Health	4.8 (1269)	6.7 (1755)

Before comparing the patterns of the risk factors within specific PHSAs (see Appendix I), more general regional patterns are described. The smoking prevalence within many of the PHSAs was considerable. Approximately 39% (7/18) of the service areas had a smoking prevalence above the provincial average of 28.3 % (Tough et al., 1999). While 61% (11/18) of the service areas had a smoking prevalence below 28.3%, only three service areas had a prevalence below 20%. PHSA #18 and Central PHSA had the highest smoking prevalences, 41.7% and 37.4 % respectively. In addition to having the highest smoking prevalence, PHSA #18 also had the largest proportion of women who consumed alcohol (9.4%) and used street drugs (7.5%).

Numerous service areas had a large proportion of women who did not attend prenatal classes and a substantial proportion of women who had less than 8 visits during their pregnancy. In 38.8% of the PHSAs (7/18), more than 60% of the women did not attend prenatal classes. Central, North Central, and PHSA #18 had the highest proportion of women who did not attend classes, 70.2%, 67.2%, and 65.5% respectively. Of the 11 service areas that had less than 60 % of the women who did not attend classes, Sherwood Park had the lowest non-attendance recorded (43.7%). There were also a number of women who had very few prenatal visits (≤ 7 visits) during their pregnancy. In one third of the PHSAs (n=6), more than 20 % of the women had seven or fewer visits during pregnancy.

Risk factors with low prevalences were low pre-pregnancy weight, poor gestational weight gain during pregnancy, and anemia during pregnancy. In 94.4% (n=17) of the PHSAs, fewer than 2% of the women had insufficient weight gain during their pregnancy. Similarly, fewer than 1% of the women within each PHSA had anemia during their pregnancy.

Two categories of risk factors that often require medical intervention are pre-existing medical diseases and complications of pregnancy. To assess the distribution of pre-existing medical diseases within the PHSAs, those women with any of the pre-existing medical diseases (heart disease, diabetes mellitus, chronic hypertension and renal disease, other medical diseases) were identified as unhealthy prior to pregnancy. Otherwise, the women were identified as healthy. Similarly, women with complications of pregnancy (gestational hypertension, pre-eclampsia, gestational bleeding) were defined as high-risk (versus low-risk if the women did not have any complications). The percentage of women who had pre-existing medical diseases ranged from a low of 4.4 % in Strathcona County to a high of 7.0 % in St. Albert. West (North), West (South) and PHSA #18 had the highest proportion of women with complications, 14.2%, 15.8 %, and 12.7 % respectively.

To better understand the geographical distribution of modifiable and non-modifiable risk factors and birth outcomes, and to address issues associated with the variations in population sizes across the PHSAs, geographic rates were calculated and PHSA maps were created. The mapping process permitted identification of those rates that did not differ significantly from CHA, those that were probably lower than CHA, those that were significantly lower than CHA, those that were higher than CHA, and those that were significantly higher than CHA. As noted in Chapter 3 (Methods), Appendix D provides a description of the calculation of the geographic rates and how to interpret the risk factor and birth outcome maps.

As noted in Figure 7, Central, North Central, West (North), Millwoods, and PHSA #18 all had a LBW rate that was higher than for the CHA. Figure 7 demonstrates that Central, West (North), and PHSA # 18 had a PTB rate that was higher or significantly higher than the overall rate of CHA. In addition to similar birth outcome patterns, Central and PHSA #18 also had several comparable risk factor prevalences. Figures 9 to 13 present the PHSA differences for smoking, use of street drugs, non-attendance at prenatal classes, prenatal visits (≤4 visits), and obstetrical history (previous PTB). These figures demonstrate that PHSA #18 and Central PHSA consistently had significantly higher rates than CHA for smoking, use of street drugs, lack of prenatal class attendance, and reduced number of prenatal visits to their health care provider. Furthermore, in comparison to CHA as a whole, both of these PHSAs had either a higher or significantly higher distribution of women who had a previous PTB.

Figure 7. Low Birth Weight Distributions Across Public Health Service Areas

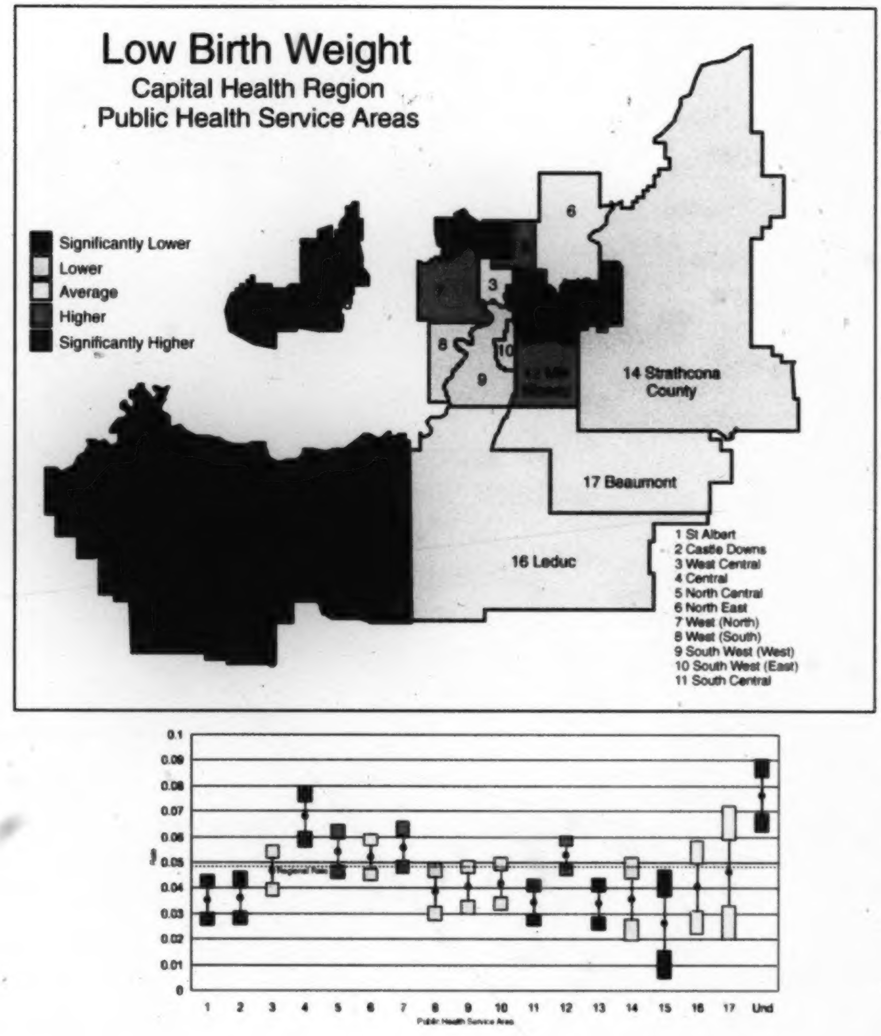
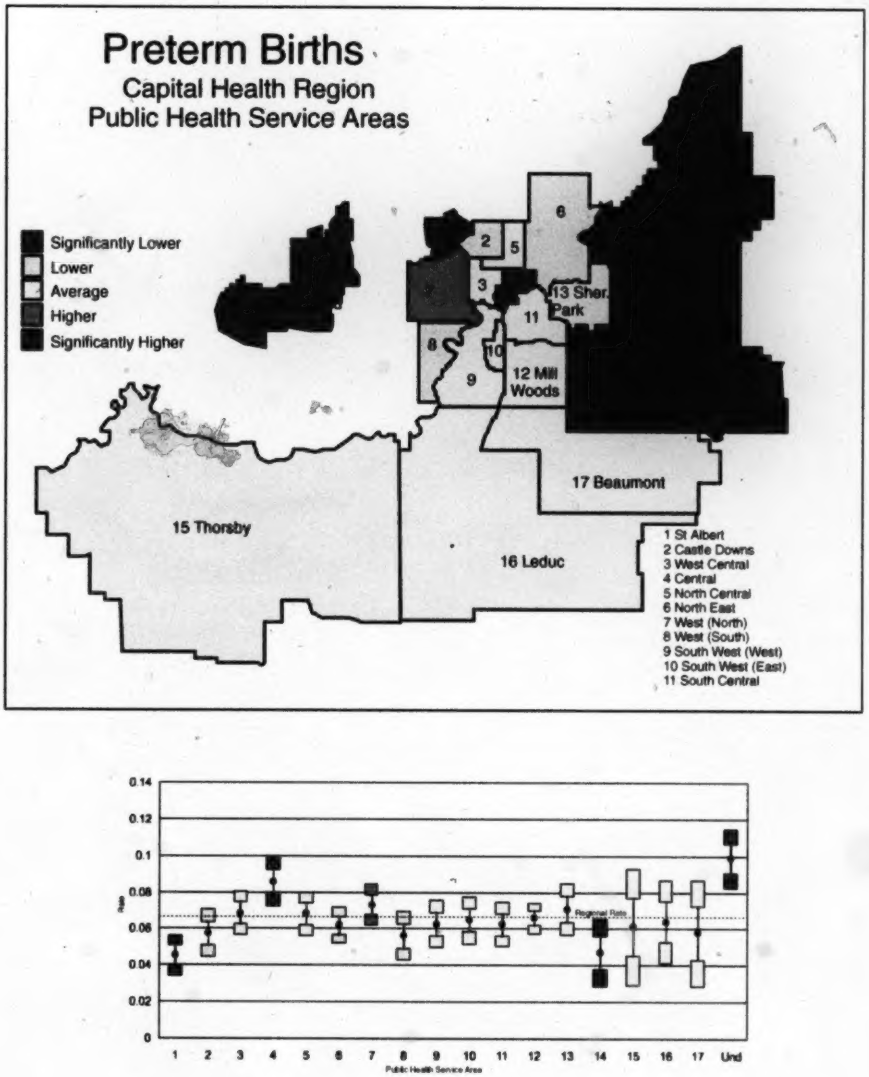

Low Birth Weight
Capital Health Region
Public Health Service Areas
Significantly Lower
Lower
Average
Higher
Significantly Higher
6
3
8
9
10
14 Strathcona County
17 Beaumont
16 Leduc
1 St Albert
2 Castle Downs
3 West Central
4 Central
5 North Central
6 North East
7 West (North)
8 West (South)
9 South West (West)
10 South West (East)
11 South Central
0.1
0.09
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
Regional Rate
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
Und
Public Health Service Area

Figure 8. Preterm Birth Distributions Across Public Health Service Areas


Preterm Births
Capital Health Region
Public Health Service Areas
Significantly Lower
Lower
Average
Higher
Significantly Higher
2
5
6
3
13 Sher. Park
11
8
10
9
12 Mill Woods
17 Beaumont
15 Thorsby
16 Leduc
1 St Albert
2 Castle Downs
3 West Central
4 Central
5 North Central
6 North East
7 West (North)
8 West (South)
9 South West (West)
10 South West (East)
11 South Central
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
Rate
Regional Rate
1
2
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4
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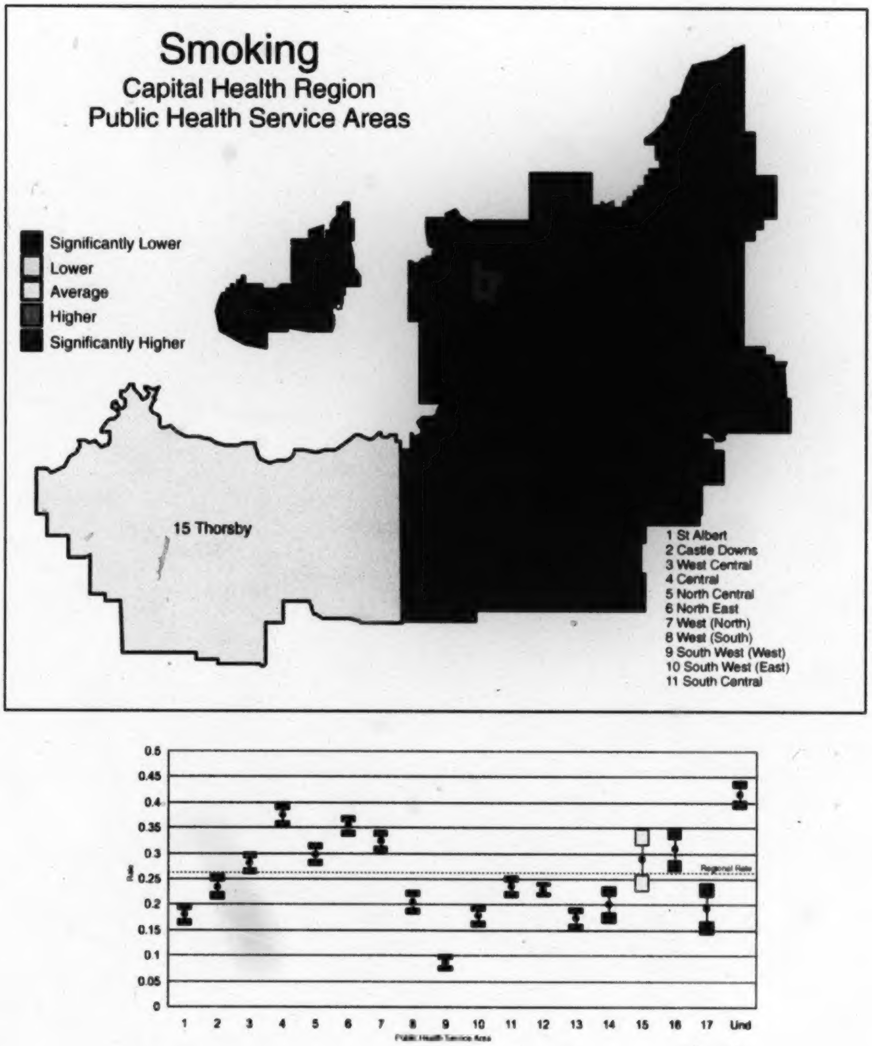
14
15
16
17
Und
Public Health Service Area

Figure 9. Public Health Service Area Differences in Smoking


Smoking
Capital Health Region
Public Health Service Areas
Significantly Lower
Lower
Average
Higher
Significantly Higher
15 Thorsby
1 St Albert
2 Castle Downs
3 West Central
4 Central
5 North Central
6 North East
7 West (North)
8 West (South)
9 South West (West)
10 South West (East)
11 South Central
0.5
0.45
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
Rate
Regional Rate
1
2
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4
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7
8
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12
13
14
15
16
17
Und
Public Health Service Area

Figure 10. Public Health Service Area Differences in Use of Street Drugs

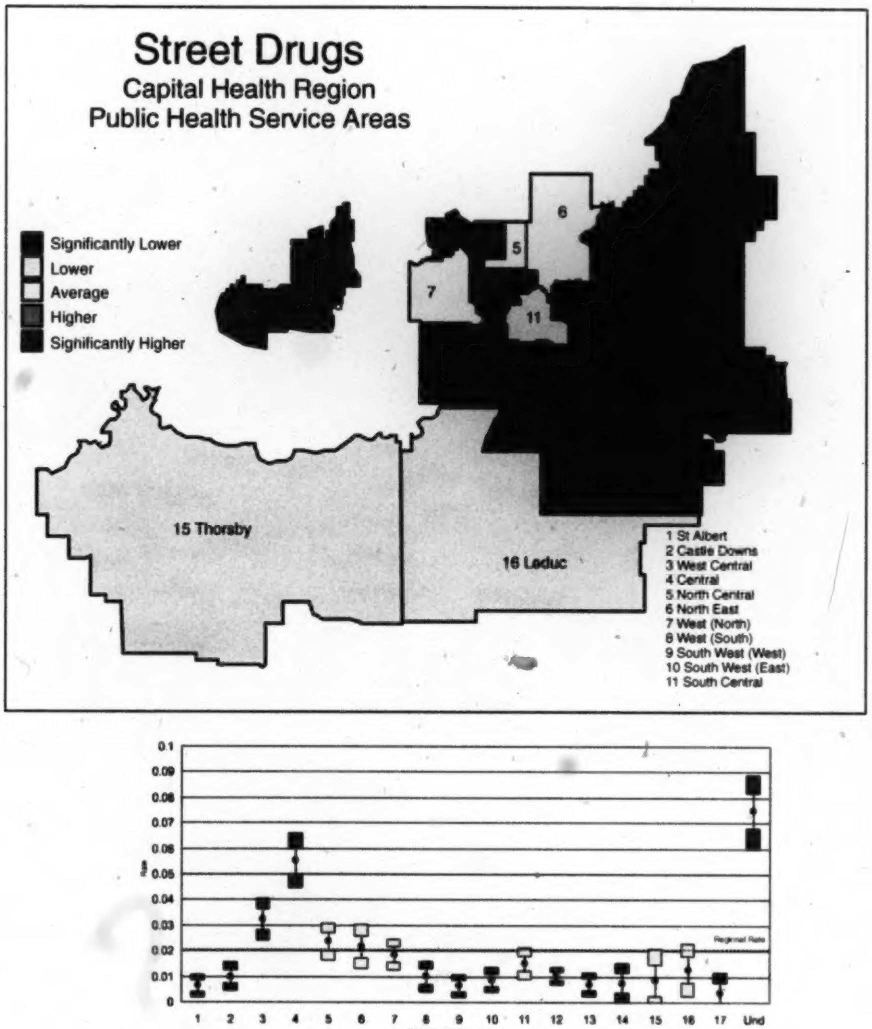

Street Drugs
Capital Health Region
Public Health Service Areas
Significantly Lower
Lower
Average
Higher
Significantly Higher
5
6
7
11
15 Thorsby
16 Leduc
1 St Albert
2 Castle Downs
3 West Central
4 Central
5 North Central
6 North East
7 West (North)
8 West (South)
9 South West (West)
10 South West (East)
11 South Central
0.1
0.09
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
Rate
Regional Rate
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
Und
Public Health Service Area

Figure 11. Public Health Service Area Differences in Prenatal Class Attendance

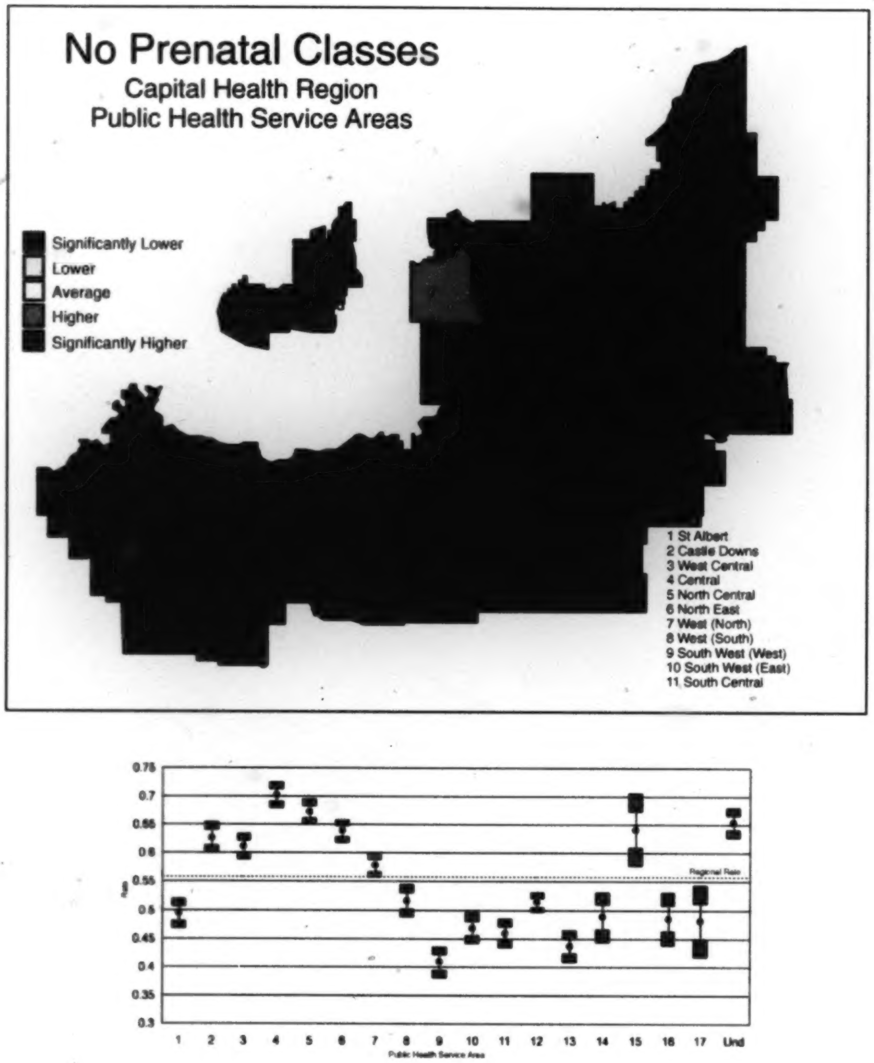


Figure 12. Public Health Service Area Differences in Prenatal Visits

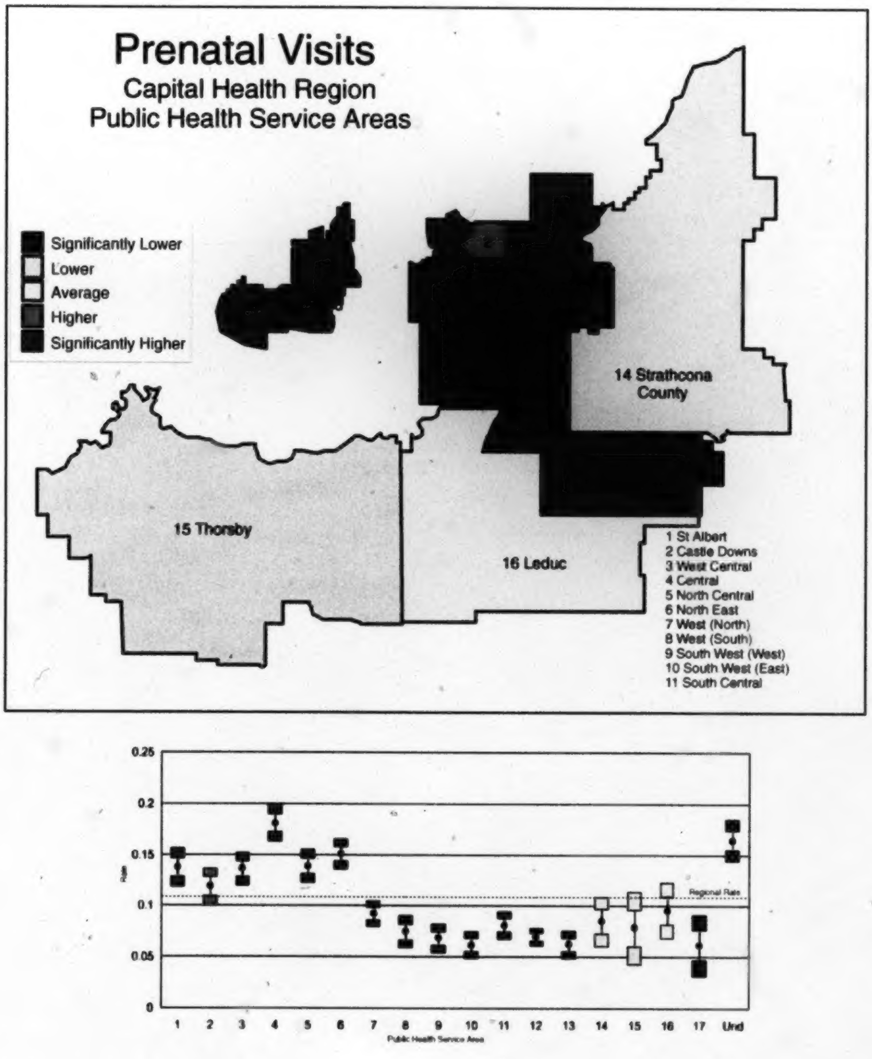
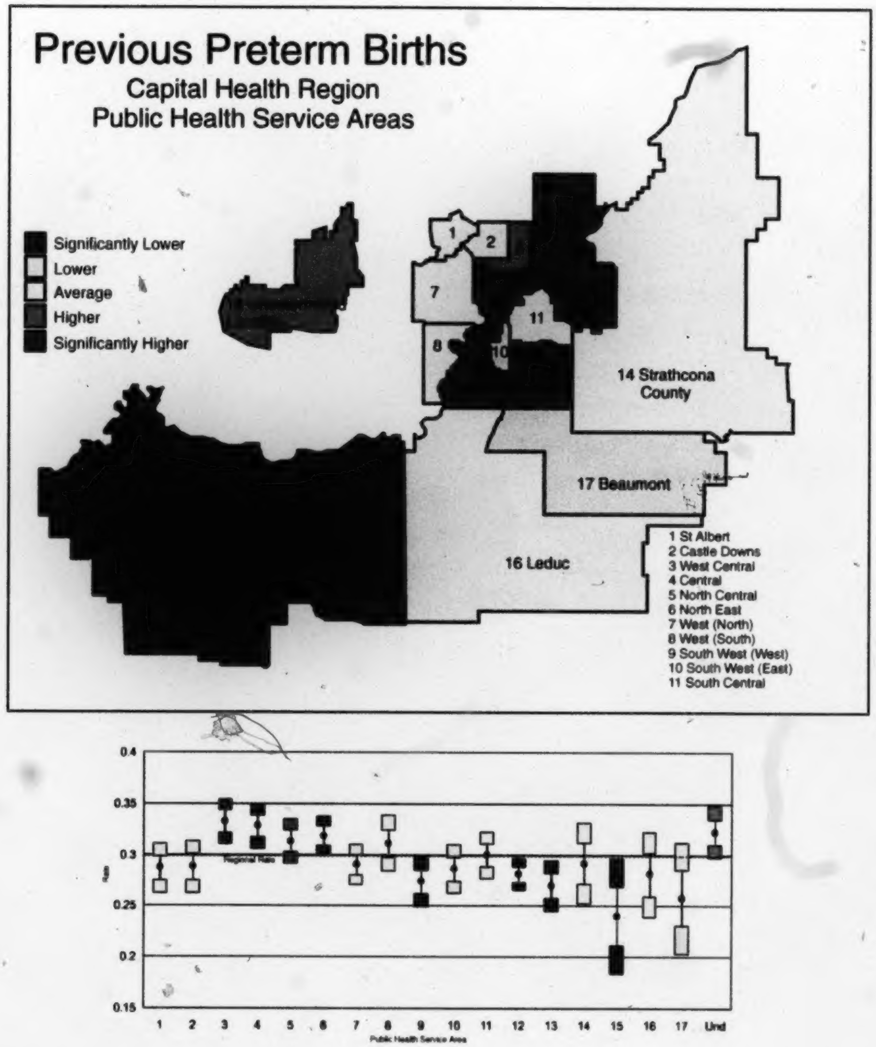

Prenatal Visits
Capital Health Region
Public Health Service Areas
Significantly Lower
Lower
Average
Higher
Significantly Higher
14 Strathcona County
15 Thorsby
16 Leduc
1 St Albert
2 Castle Downs
3 West Central
4 Central
5 North Central
6 North East
7 West (North)
8 West (South)
9 South West (West)
10 South West (East)
11 South Central
0.25
0.2
0.15
0.1
0.05
0
Regional Rate
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
Und
Public Health Service Area

Figure 13. Public Health Service Area Differences in Previous Preterm Births


Previous Preterm Births
Capital Health Region
Public Health Service Areas
Significantly Lower
Lower
Average
Higher
Significantly Higher
1
2
7
11
8
10
14 Strathcona County
17 Beaumont
16 Leduc
1 St Albert
2 Castle Downs
3 West Central
4 Central
5 North Central
6 North East
7 West (North)
8 West (South)
9 South West (West)
10 South West (East)
11 South Central
0.4
0.35
0.3
0.25
0.2
0.15
Regional Rate
Rate
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
Und
Public Health Service Area

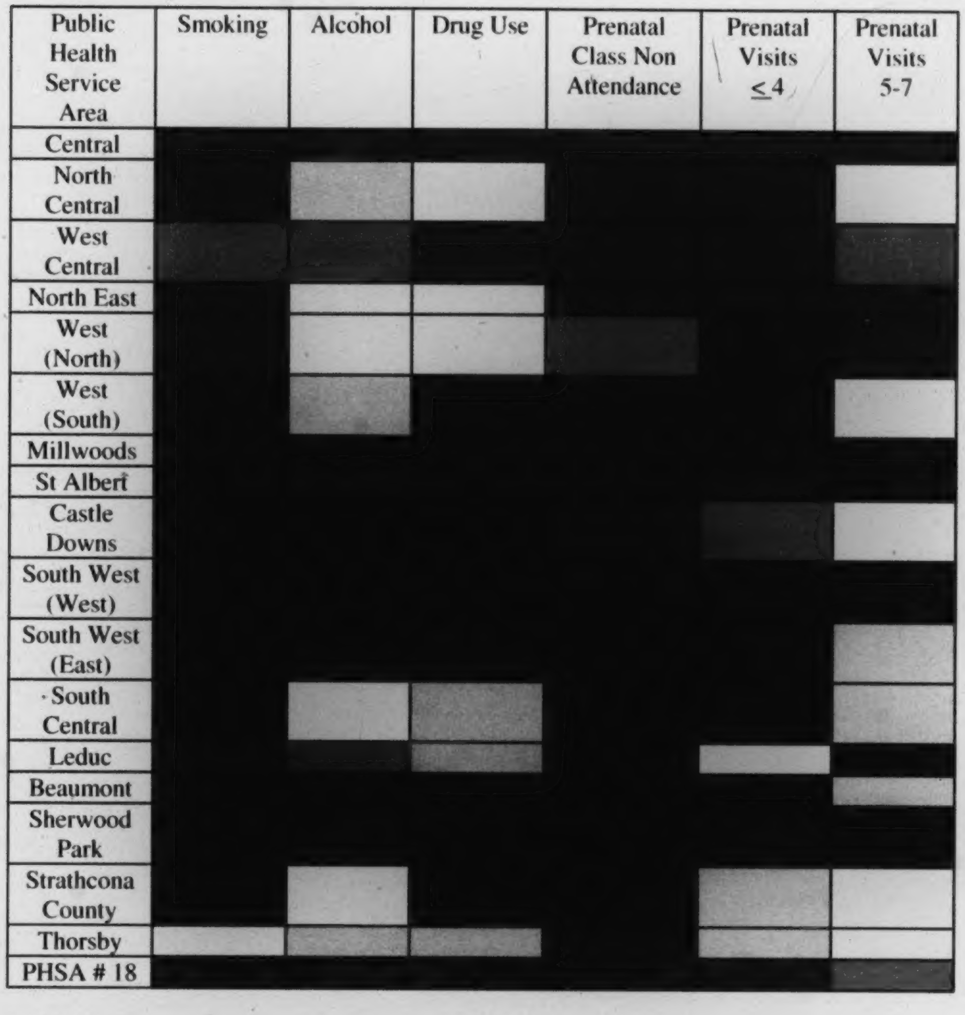
There were several PHSAs (South West West (SWW), South West East (SWE), West South, Leduc, Beaumont, Strathcona) where the LBW and PTB rates were either average (not likely to differ from CHA) or lower than CHA. Although these PHSAs did not have identical risk factor prevalences, the overall distributions of the risk factors were predominantly identified as average, lower, or significantly lower than CHA. For example, the LBW rate for SWW and SWE was below that of CHA. The risk factor prevalences of prenatal care, lifestyle behaviors, pregnancy complications, pre-existing medical diseases, obstetrical history, and medical problems of pregnancy for these PHSAs were also either average or below CHA. The only exceptions to this pattern were in SWE where the prevalence of poor gestational weight gain was higher than CHA and in West South, where several pregnancy complications were all higher or significantly higher than CHA. For SWW, SWE, West South, South Central, Beaumont, Thorsby, Leduc, Sherwood Park, and Strathcona County, the risk factor prevalences that were higher or significantly higher than CHA were predominantly isolated to either one risk factor grouping (i.e., pregnancy complications, lifestyle factors, prenatal care), or one or two risk factors within the risk factor groupings (i.e., smoking, previous SGA, anemia, poor gestational weight gain, polyhydramnios /oligohydramnios).

One PHSA (North East) did not present with a LBW or a PTB rate higher than CHA, yet it appeared to have a high prevalence of several risk factors. In North East, with the exception of pregnancy complications, several of the lifestyle and prenatal care risk factors were significantly higher than CHA, several obstetrical risk factors (previous PTB, previous SGA birth) were higher or significantly higher than CHA, and several of the pre-existing medical diseases and medical problems during pregnancy (chronic hypertension, diabetes, polyhydramnios/oligohydramnios, and acute medical problems) were also all higher than CHA.

To provide a visual display of the risk factor prevalences within the PHSA, each individual map was color-coded in a table to provide a summary of a selected grouping of risk factors. The white coding within the table indicates there were no cases in the PHSA (Edwards, Schopflocher, Fraser-Lee, 2000). Lifestyle and prenatal care factors are represented in Table 10, pregnancy complications in Table 11, obstetrical history in Table 12, nutritional problems during pregnancy in Table 13, medical problems during pregnancy in Table 14, and pre-existing medical diseases in Table 15.

It was not apparent from the maps or the tables that those PHSAs with a LBW rate or a PTB rate higher than CHA also had higher prevalences than CHA for ***each*** specific risk factor that was predictive in the LBW or PTB model. Nor was it evident that PHSAs that had PTB or LBW rates that were the same or below CHA also had lower risk factor prevalences for ***all*** factors predictive of either birth outcome. From review of the maps and tables we can say that: a) both Central and PHSA #18 had a significantly higher LBW rate and PTB rate than CHA; b) the majority of risk factors in Central and PHSA #18 were either higher or significantly higher than CHA; c) both Central and PHSA #18 had a strong presence of a number of the risk factors that were predictive of both birth outcomes; d) the ***overall*** pattern of the remaining PHSAs was relatively homogeneous in both risk factors and outcomes; and e) although the maps allowed for comparison of the PHSAs, the degree of simplification required by the mapping and tabularization suggests that not all aspects of the models could be captured by such gross measures. Therefore, it was neither possible nor appropriate to make causal inferences about the differential impact of geography on risk factor prevalences and birth outcomes. However, it should be noted that Central PHSA is a more disadvantaged area in terms of the variables used to reflect SES (see Appendix A). This PHSA had a high prevalence of women with poor birth outcomes and a high prevalence of risk factors predictive of PTB and LBW.

Table 10
Lifestyle and Prenatal Care Factors

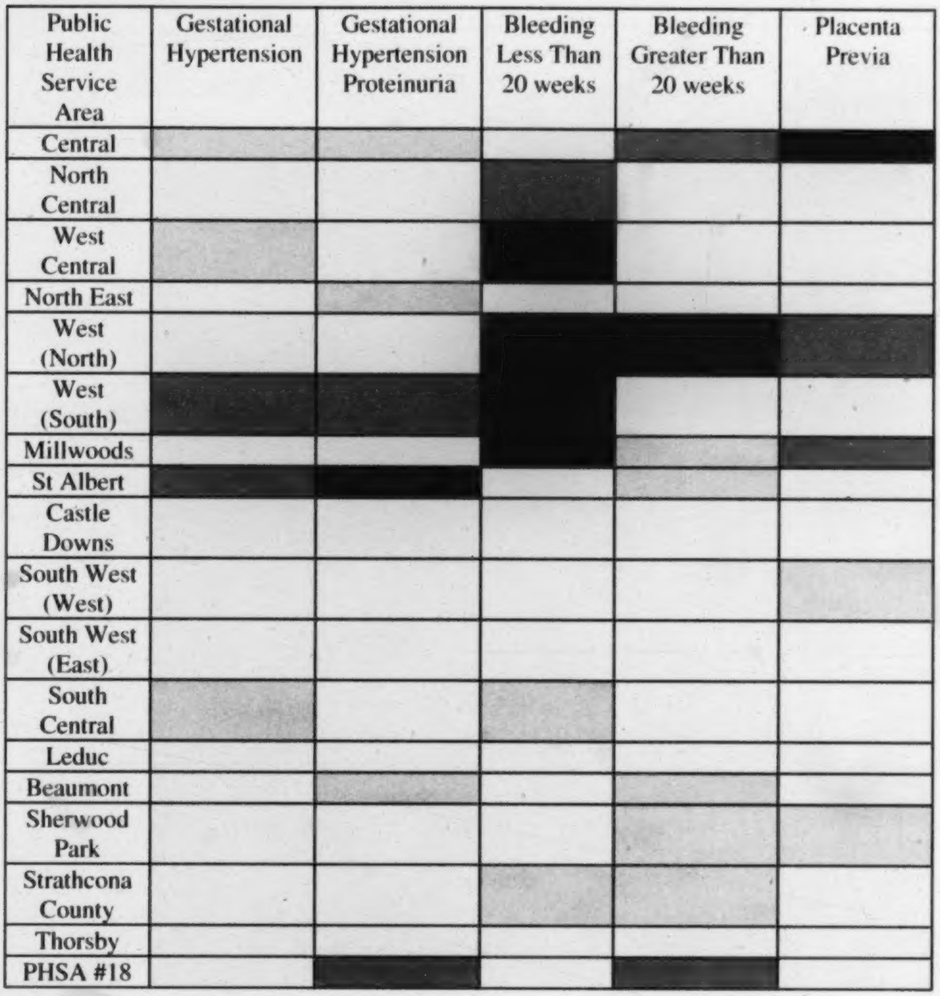


Public Health Service Area	Smoking	Alcohol	Drug Use	Prenatal Class Non Attendance	Prenatal Visits ≤4	Prenatal Visits 5-7
Central	■	■	■	■	■	■
North Central	■	▒	□	■	■	□
West Central	▓	▓	■	■	■	▓
North East	■	□	□	■	■	■
West (North)	■	□	□	▓	■	■
West (South)	■	▒	■	■	■	□
Millwoods	■	■	■	■	■	■
St Albert	■	■	■	■	■	■
Castle Downs	■	■	■	■	▓	□
South West (West)	■	■	■	■	■	■
South West (East)	■	■	■	■	■	▒
South Central	■	□	▒	■	■	▒
Leduc	■	▓	▒	■	□	■
Beaumont	■	■	■	■	■	▒
Sherwood Park	■	■	■	■	■	■
Strathcona County	■	□	■	■	▒	□
Thorsby	□	▒	▒	■	□	□
PHSA # 18	■	■	■	■	■	▓

Legend

■	Significantly Lower
▒	Lower
□	Average
▓	Higher
■	Significantly Higher

Table 11
Pregnancy Complications

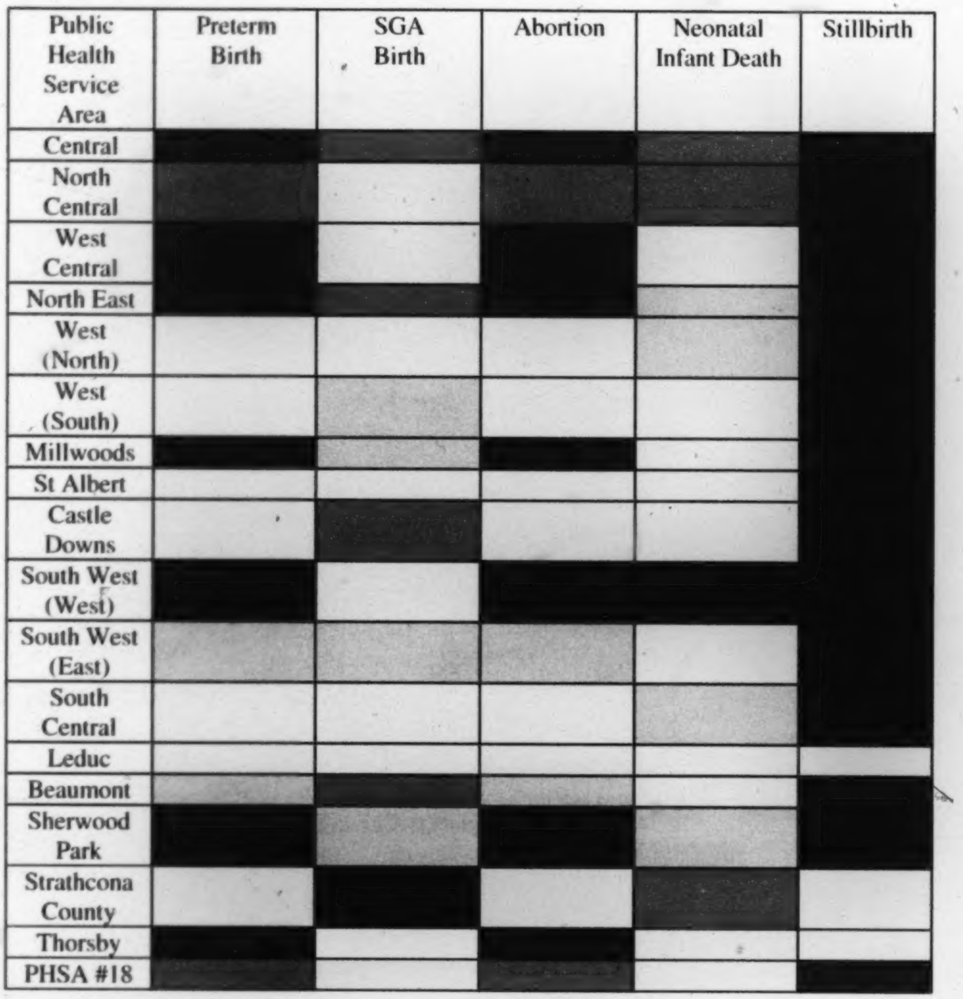


Public Health Service Area	Gestational Hypertension	Gestational Hypertension Proteinuria	Bleeding Less Than 20 weeks	Bleeding Greater Than 20 weeks	Placenta Previa
Central	▨	▨	□	▩	■
North Central	□	□	▩	□	□
West Central	▨	□	■	□	□
North East	□	▨	▨	□	□
West (North)	□	□	■	■	▩
West (South)	▩	▩	■	□	□
Millwoods	□	□	■	▨	▩
St Albert	▩	■	□	▨	□
Castle Downs	□	□	□	□	□
South West (West)	□	□	□	□	▨
South West (East)	□	□	□	□	□
South Central	▨	□	▨	□	□
Leduc	□	□	□	□	□
Beaumont	□	▨	□	▨	□
Sherwood Park	□	□	□	▨	▨
Strathcona County	□	□	▨	▨	□
Thorsby	□	□	□	□	□
PHSA #18	□	▩	□	▩	□

Legend

■	Significantly Lower
▨	Lower
□	Average
▩	Higher
■	Significantly Higher

Table 12
Obstetrical History



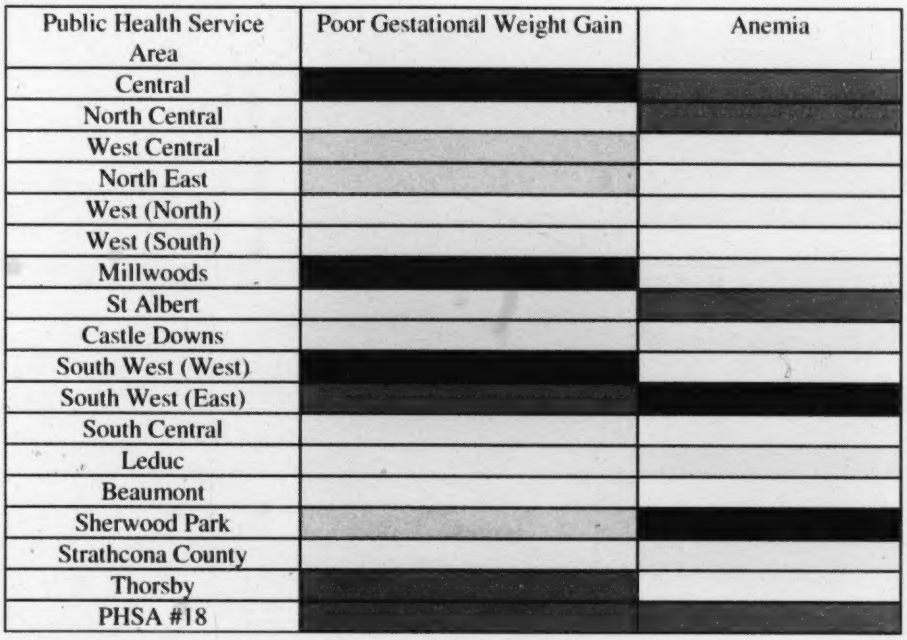
Public Health Service Area	Preterm Birth	SGA Birth	Abortion	Neonatal Infant Death	Stillbirth
Central	■	▓	■	▓	■
North Central	▓	□	▓	▓	■
West Central	■	□	■	□	■
North East	■	▓	■	▒	■
West (North)	□	□	□	▒	■
West (South)	□	▒	□	□	■
Millwoods	■	▒	■	□	■
St Albert	□	□	□	□	■
Castle Downs	□	▓	□	□	■
South West (West)	■	□	■	■	■
South West (East)	▒	▒	▒	□	■
South Central	□	□	□	▒	■
Leduc	□	□	□	□	□
Beaumont	▒	▓	▒	□	■
Sherwood Park	■	▒	■	▒	■
Strathcona County	□	■	□	▓	□
Thorsby	■	□	■	□	□
PHSA #18	▓	□	▓	□	■

Legend

■	Significantly Lower
▒	Lower
□	Average
▓	Higher
■	Significantly Higher

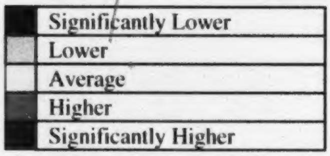
Table 13

Nutritional Problems During Pregnancy



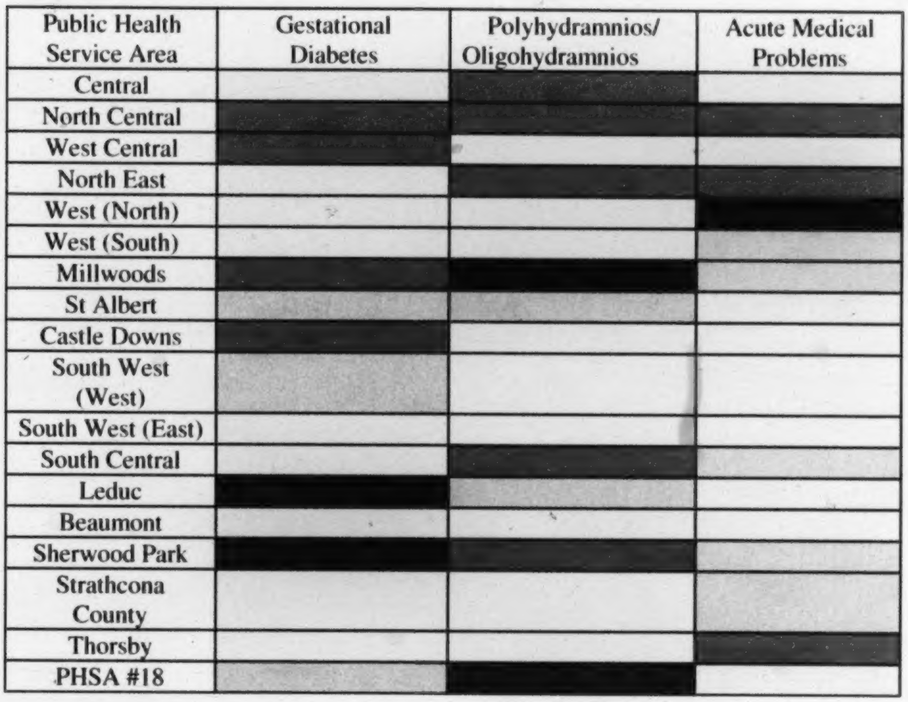
Public Health Service Area	Poor Gestational Weight Gain	Anemia
Central	■	▓
North Central	□	▓
West Central	▒	□
North East	▒	□
West (North)	□	□
West (South)	□	□
Millwoods	■	□
St Albert	□	▓
Castle Downs	□	□
South West (West)	■	□
South West (East)	▓	■
South Central	□	□
Leduc	□	□
Beaumont	□	□
Sherwood Park	▒	■
Strathcona County	□	□
Thorsby	▓	□
PHSA #18	▓	▓

Legend



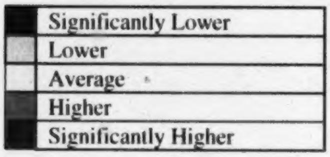
■	Significantly Lower
▒	Lower
□	Average
▓	Higher
■	Significantly Higher

Table 14
Medical Problems During Pregnancy



Public Health Service Area	Gestational Diabetes	Polyhydramnios/ Oligohydramnios	Acute Medical Problems
Central	□	▓	□
North Central	▓	▓	▓
West Central	▓	□	□
North East	□	▓	▓
West (North)	□	□	■
West (South)	□	□	▒
Millwoods	▓	■	▒
St Albert	▒	▒	□
Castle Downs	▓	□	□
South West (West)	▒	□	□
South West (East)	□	□	□
South Central	□	▓	▒
Leduc	■	▒	□
Beaumont	▒	□	□
Sherwood Park	■	▓	▒
Strathcona County	□	□	▒
Thorsby	□	□	▓
PHSA #18	▒	■	□

Legend



■	Significantly Lower
▒	Lower
□	Average
▓	Higher
■	Significantly Higher

Table 15

Pre-existing Medical Diseases

Public Health Service Area	Heart Disease	Diabetes Mellitus	Renal Disease	Chronic Hypertension	Other Medical Diseases
Central	□	□	▓	□	□
North Central	■	▓	□	▓	□
West Central	□	■	□	□	□
North East	□	▓	□	▓	░
West (North)	□	░	□	□	▓
West (South)	□	■	□	□	□
Millwoods	░	□	░	░	░
St Albert	▓	■	□	▓	▓
Castle Downs	□	▓	□	□	□
South West (West)	□	□	□	□	□
South West (East)	■	□	□	□	□
South Central	□	░	□	■	░
Leduc	□	░	□	□	□
Beaumont	□	□	□	░	□
Sherwood Park	□	■	□	□	░
Strathcona County	□	░	□	░	■
Thorsby	▓	□	□	□	□
PHSA #18	▓	□	▓	▓	□

Legend

■	Significantly Lower
░	Lower
□	Average
▓	Higher
■	Significantly Higher

Chapter 6 Discussion

In this final chapter, the findings relevant to the risk factors are discussed within the context of the research literature. The strengths and limitations of the study are then addressed. The chapter concludes with the implications for nursing practice, policy and program development, and suggestions for future research.

The primary objectives of this study were to describe the distribution of birth outcomes (PTB and LBW) and risk factors across the PHSAs of CHA and to identify the direct and indirect effect of these risk factors on birth weight and gestational age. To increase understanding of the effect of these potential risk factors on the study birth outcomes, a hypothetical model was developed to guide the analyses. The premises of this model were: a) there are distal (e.g., maternal age) and proximal (e.g., pregnancy complications) risk factors for LBW and PTB; and, b) risk factors can have a direct (independent) effect and/or indirect effect on birth outcomes (e.g., the effect of a more distal risk factor (maternal age) is mediated by (operates through) more proximal risk factors (prenatal care or pregnancy complications)).

Four models were developed: two models for PTB and two models for LBW. One of the LBW models and one of the PTB models included the aggregate SES factors. These models allowed for determination of the direct and indirect effects of risk factors on PTB and LBW.

Discussion of Risk Factors Associated with LBW and PTB

Maternal Age

Maternal age was not a significant predictor for PTB or LBW. However, maternal age indirectly affected birth weight and gestational age through its influence on other risk factors. Younger women were less likely to receive “adequate” prenatal care (8 or more visits and attend prenatal classes), and were therefore at greater risk for delivering a preterm or LBW infant. Older women were more likely to develop pregnancy complications, and were therefore more at risk for delivering early, most likely as a result of medical intervention. Consequently, these women were at greater risk of having a preterm or LBW infant.

The study finding that young maternal age does not exert a direct effect on adverse birth outcomes when age-related confounders are taken into consideration is consistent with several other investigations (Berkowitz, 1985; Berkowitz & Papiernik, 1993; Ekwo & Moawad, 2000; Kramer et al., 1992, Lang et al., 1996; Meis, Goldenberg, Mercer, Iams, Moawad, & Miodovnik, 1998). The association between young maternal age and birth outcomes may be related to how the birth outcomes (i.e., LBW and PTB) were operationalized. Michielutte et al. (1992), controlling for age-related confounders (i.e., education, smoking, inadequate prenatal care, low-pre-pregnancy weight) found that younger women had an increased risk for delivering a preterm LBW infant. However, there was no association between young maternal age and LBW when the LBW was operationalized as term LBW infants or all LBW infants (regardless of gestational age). Similarly, when Meis et al. (1995) examined separate PTB models, they found a positive association between younger maternal age and spontaneous PTB but not for medically indicated PTBs after controlling for age -dependent confounders (i.e., smoking, pre-pregnancy weight and height, SES).

The null finding in this study of no direct effect of older maternal age on PTB is also consistent with several previous studies (Kramer et al., 1992; Lang et al., 1996). Again, the research findings regarding the effect of older maternal age on PTB differ according to the operationalization of PTB, the control for relevant confounders, and the other risk factors examined in the study. For example, Meis et al. (1995) found an increased risk for medically indicated PTBs but not for spontaneous PTBs for older women, whereas Berkowitz et al. (1998) reported an increased risk for preterm premature rupture of membranes but not for preterm labor or medically induced PTB. Both teams of investigators controlled for SES, smoking, and pre-pregnancy weight measures; however, they differed in the medical problems, pregnancy complications, and the obstetrical risk factors examined. The findings of these investigators suggest that different medical problems and pregnancy complications have different influences on the pregnancy, resulting in different outcomes (i.e., preterm labor, medically indicated preterm births).

In summary, the disparate research findings regarding maternal age are congruent with the suggestion of Berkowitz and Papiernik (1993) that study differences may be a result of variations in the risk factors examined across the different studies, control of confounding variables, and the operationalization of the birth outcomes. This study controlled for age-related confounders and a number of other risk factors, and is consistent with other investigators who have suggested that maternal age is not predictive of PTB or LBW (Kramer et al., 1992; Meis et al., 1998). This study is unique because the potential mechanisms by which maternal age affects gestational age and birth weight are identified.

There is no consensus among experts whether maternal chronological age is an *independent* determinant of either fetal growth or gestational duration, or whether the age of the mother may be a risk marker for lifestyle, psychosocial problems, economic, and medical circumstances. Consequently, it has been suggested that maternal age *indirectly* impacts birth weight and/or gestational duration (Fraser et al., 1995; Jimenez, Martin, & Garcia, 2000; Jolly et al., 2000). This study found that maternal age exerts an indirect effect on birth weight and gestational age, but more importantly, the results provide insight about the factors through which maternal age operates (i.e., SES, prenatal care, pregnancy complications).

Obstetrical History: Parity, Previous Preterm or SGA Birth

In this study, nulliparous women were at greater risk of having a PTB. This finding is consistent with the findings of several other investigators (Lang et al., 1996; Meis et al., 1995). It was shown in this study that the influence of nulliparity on LBW was through its effect on gestational age.

A woman's past obstetrical history (e.g., previous PTB, previous SGA birth) also plays an important role in the prediction of subsequent pregnancy outcomes. Consistent with the findings of other studies, women in this study who had a previous PTB were at an increased risk for both PTB (Ancel et al., 1999; Berkowitz et al., 1998; Lang et al., 1996; Wen et al., 1990) and LBW (Kristensen, Langhoff-Roos, & Kristensen, 1995). The joint comparison of the PTB and LBW analyses further demonstrates the important effect of a previous PTB on gestational duration and the significant influence of the gestational age of the infant on LBW. Women who had a previous PTB had a 4-fold risk of a PTB, whereas in the LBW analysis, women who had a previous PTB had a 2-fold risk of having a LBW infant when gestational age was added to the model. This finding also suggests that reported associations between previous PTB and LBW may partially be a reflection of the impact of gestational duration on LBW. Researchers such as Kramer (1987) and Lumley (1993) expressed some uncertainty whether it is the actual occurrence of a previous PTB that results in a subsequent PTB. They suggest that a subsequent PTB may be due to the existence of the same risk factors in the current pregnancy. Although it was not possible in this study to determine if the same risk factors existed in both pregnancies, the strength of this study was the ability to examine a number of potential risk factors concurrently, including the adjustment of potential covariates.

Women in the study who had a previous SGA birth were not at increased risk for a PTB. However, these women were at increased risk of delivering a LBW infant. This finding suggests women who deliver a LBW infant are at an increased risk for delivering a LBW infant in a subsequent pregnancy.

Prenatal Care

Prenatal Visits. Approximately 30% of the women in this study had fewer than 8 visits to their health care provider. Women who had 4 or fewer prenatal visits demonstrated a 4-fold risk for PTB, and a 2-fold risk for delivering a LBW infant. This finding is consistent with Tough et al. (1999) who reported that women with fewer than four visits had almost a 3-fold risk of delivering a preterm infant or LBW infant. Sable and Herman (1997), Kogan et al. (1994), Ekwo and Moawad (2000), and Collins, Wall and David (1997) also reported increased risks for LBW (ORs ranging from 1.3 to 7.2) for women who had inadequate prenatal care. These studies were not consistent in their definition of inadequate prenatal care. Inadequate care was defined as late initiation of prenatal care (e.g., 12 weeks gestation), insufficient content of prenatal care (e.g., health behavior advice and procedures performed), or inadequate utilization of care (e.g., an algorithm derived from number of visits, trimester prenatal care begun, and adjusted visits for gestational age at delivery).

Prenatal visits have been endorsed both as a means to identify mothers at risk for delivering a preterm or growth retarded infant, and also as an opportunity to provide an array of medical, nutritional, and lifestyle interventions intended to reduce the determinants of LBW and its overall incidence (Alexander & Korenbrot, 1995; Petersen, Connelly, Martin, & Kupper, 2001). In this study, a reduced number of prenatal visits (7 or fewer visits) was associated with both PTB and LBW. It is possible that the reduced number of visits was inadequate to ensure that the woman received the benefits associated with primary prevention strategies (i.e., promoting a healthy lifestyle and avoiding or reducing risk factors so that preterm labor or premature rupture of membranes does not occur) and secondary prevention strategies (i.e., identification of women at risk through risk assessment, early detection of pregnancy complications or medical conditions).

Prenatal Classes. Prenatal classes are an alternate means to provide counselling related to healthy eating, smoking cessation, stress reduction, and the signs and symptoms of complications such as premature rupture of membranes and preterm labor (Davies et al., 1998). If women are unable to attend classes, then they may have missed opportunities for information about risks and risk reduction strategies (McCormick & Siegel, 2001; Prozialeck & Pesole, 2000). In this study, in more than one third of the PHSAs (39%) more than 60% of the women did not attend prenatal classes. Furthermore, all of the socially disadvantaged PHSAs (i.e., Central, North Central and West Central) had a significantly higher prevalence of women who did not attend prenatal classes. Women who did not attend classes had an increased risk of delivering a preterm infant. On the other hand, only nulliparous women who did not attend prenatal classes were at greater risk for delivering a LBW infant.

Although childbirth education classes include preventive and health promotion counselling regarding lifestyle issues and complications of pregnancy, research has not evaluated if mothers adopted the information provided in the classes (Libbus & Sable, 1991; Lynch & Young, 1997; Sturrock & Johnson, 1990). Furthermore, to my knowledge, researchers have not investigated the effects of prenatal childbirth classes on infant outcomes. Rather, the childbirth education literature has primarily focused on labor and postpartum outcomes such as breastfeeding, pain during labor, coping, and parenting (Handfield & Bell, 1995; Slaninka, Galbraith, Strzelecki, & Cockcroft, 1996), and obstetrical outcomes such as length of labor and presence of lacerations (Hetherington, 1990; Sturrock & Johnson, 1990). Consequently, comparison of the study findings with the research literature was not possible.

Lifestyle Risk Factors

The increased incidence of LBW and PTB among women who smoke has been well documented for more than two decades. Nevertheless, a significant proportion of women continue to smoke during pregnancy (Adams & Melvin, 1998; Cooke, 1998; England et al., 2001; Kolas, Nakling, & Salvensen, 2000; Kyrklund- Blomberg & Cnattingius, 1998; Moore & Zaccaro, 2000). In this study, one quarter (26.2%) of women resident in CHA smoked during their pregnancies. Moreover, 39% of the PHSAs in CHA had smoking prevalences during pregnancy above the provincial average of 28.3% (Tough et al., 1999), and all socially disadvantaged PHSAs had smoking prevalences that were higher than the CHA prevalence. The smoking prevalence in CHA and in many of the PHSAs was higher than that reported for Canada (21.3% to 23%) and the US (20.6% to 23%) (Adams & Melvin, 1998; Best Resource Centre, 1998; Canadian Perinatal Surveillance System Steering Committee, 2000; DiClemente, Dolan-Mullen, & Windsor, 2000; Kolas et al., 2000; Orleans, Johnson, Barker, Kaufman, & Marx, 2001; Siener, Malrcher, & Husten, 2000).

Smoking was not a significant predictor of PTB in this study. The consistency of this study finding with other studies (Ancel et al, 1999; Horta et al, 1997; Meis et al., 1998;Wen et al., 1990) appears to be dependent on how smoking status was defined (i.e., smoked during pregnancy, smoked 1 to 9 cigarettes per day, smoked 10 or more cigarettes per day), and how PTB was operationalized (i.e., spontaneous or medically indicated PTBs, preterm birth defined as less than 37 weeks gestation). Meis et al.

(1995) did not find an increased risk of spontaneous or medically indicated PTB for women who smoked 9 or fewer cigarettes a day. Ancel et al. (1999) also did not find an increased risk for spontaneous PTBs, however they did report an increased risk for medically indicated PTBs. These findings suggest that smoking during pregnancy may be associated with specific pathways that lead to PTB. In this study it was not possible to separate out the effects of smoking on different PTB pathways.

Although smoking was not a significant predictor for PTB, further analyses in this study revealed that the impact of smoking on PTB may be through its effects on gestational bleeding. Researchers have found that women who smoked during pregnancy had almost a three–fold risk of pregnancy complications such as placenta previa and gestational bleeding. These complications can lead to spontaneous preterm delivery or medically indicated induced preterm delivery (Ananth et al., 2001; Cnattingius et al., 1997; Odendaal et al., 2001).

In contrast to the PTB findings, women in this study who smoked had a 2-fold risk of delivering a LBW infant. A number of investigators agree that preterm delivery accounts for some of the LBW associated with smoking (Bonellie, 2001; Bouchkaert, 2000; Floyd, Rimer, Giovino, Mullen, & Sullivan, 1993). However, the primary impact of smoking on LBW is on fetal growth (Bonellie, 2001; Bouchkaert, 2000; Floyd et al., 1993; Leiberman, Gremy, Lang, & Cohen, 1994; Secker-Walker, et al., 1998). Estimates of the relative risk for LBW among women who smoke ranged from 1.8 to 4.0 (Abel, 1997; Horta et al., 1997; Michielutte et al., 1992; Windham et al., 2000), risks congruent with the increased risk found in this study (OR= 2.02).

Increasing drug and alcohol use by pregnant women in recent years also continues to be reported in the literature (Sweeney, Schwartz, Mattis, & Vohr, 2000). Prevalences of moderate alcohol consumption range from 5% to 20% (Canadian Perinatal Surveillance System Steering Committee, 2000; Gladstone, Levy, Nulman, & Koren, 1997; Stewart, Potter, Dulberg, Niday, Nimrod, & Tawagi, 1995; Tough et al., 1999). Consistent with national trends, in this study population, 4.6 % of women in CHA consumed alcohol during pregnancy, whereas in a recent provincial study, 7.5% of the women reported the consumption of alcohol during pregnancy (Tough et al.). The non-significant findings in this study of the effect of alcohol on PTB confirm the findings of Kramer (1987), Wen et al. (1990), Kesmodel et al. (2000) and Windham et al. (2000). Kramer (1997) also noted that most studies have not found an association between consumption of small amounts of alcohol and birth weight. Kramer's conclusion and the study findings are consistent with the results of other studies that indicate that women who consume alcohol during their pregnancy do not have an increased risk of delivering a LBW infant (Horta et al., 1997; Windham et al., 1995).

While smoking and alcohol did not have direct effects on PTB, there was a joint effect. Women who smoked and consumed alcohol during pregnancy had a 2-fold risk of delivering a preterm infant. The interaction of smoking and alcohol on gestational age was further demonstrated with the substantial decrease in the OR (risk estimate) after the addition of gestational age to the LBW model. This change in OR (risk estimate) reflects the influence of smoking and alcohol on LBW through gestational age. Studies that have found a joint effect of smoking and alcohol on birth weight have not included gestational duration in the LBW analysis; thus they have reported only a significant direct effect (Sokul, Miller & Reed, 1980; Windham et al., 1995)

The prevalence of street drug use in the current study was 2.1%. Other Alberta studies have reported similar street drug use, ranging from 1.4% to 1.9% (Newburn-Cook et al., 2002; Tough et al., 1999). In Canada, illicit drug use rates among pregnant women have been reported to be as high as 20% (Field, 2000; Koren & Klien, 1997). Reports of illicit drug use in the United States have been reported in the range of 0.4 % to 27% (Bada, Das, Bauer, Shankaran, Lester, & Wright et al., 2002; Matera, Waren, Moomgy, Fink, & Fox, 1990). In the current study population, street drug use had a significant direct effect on PTB and an indirect effect on LBW through gestational age. However, when SES was included in the risk modeling, street drug use only had a significant direct effect on LBW. There were only two studies that examined the effect of general street drug or illicit drug use on gestational duration or birth weight (Chasnoff, Burns, Schnoll & Burns, 1985; Tough et al., 1999); however, only Tough et al. reported odds ratios.

Tough et al. reported an odds ratio of 1.3 for street drug use and PTB and odds ratio of 1.51 for LBW. The confidence intervals were not reported. Chasnoff et al. (1985) on the other hand reported an increased prevalence of LBW infants among women who used street drugs.

Other investigators have examined the pregnancy outcomes of women who used specific street drugs such as cocaine, heroin, and marijuana during pregnancy (Dinsmoor et al., 1994; Eyler, Behnke, Conlon, Woods, & Wobie, 1998; Lang et al., 1996; Miller & Boudreaux, 1999; Shiono et al., 1995). The conclusions drawn from a synthesis of the literature by Kramer (1987) and Berkowitz and Papiernik (1993), as well as studies by Lang et al. (1996) and Shiono et al. (1995), indicate that there is little evidence that marijuana use affects PTB. Shiono et al. did not report an increased risk of PTB or LBW for women who used cocaine, whereas Miller and Boudreaux (1999) did report an increased risk for PTB (OR=2.7) but not for LBW. Eyler et al. (1998) found that the average cocaine use per day for trimesters one and three and for the entire pregnancy was negatively related to birth length and head circumference but not to gestational age. Dinsmoor et al. reported that 45% of patients with preterm labor had evidence of recent substance abuse, in the form of a positive urine toxicologic screen, with 70% containing evidence of cocaine use. Both Berkowitz and Papiernik (1993) and Shioni et al. (1995) in their research investigations suggest that the lack of consistent and conclusive associations between street drug use and adverse birth outcomes is due to methodological limitations such as small sample size, lack of control of confounders, and operationalization of street drug use (i.e., cocaine use, marijuana, illicit drug use).

Nutritional and Constitutional Risk Factors During Pregnancy

In this study, very few women had a poor pre-pregnancy weight (0.4%), poor gestational weight gain (1.1%), or anemia (0.3%) during pregnancy. Poor gestational weight gain during pregnancy was not a predictor for PTB. The study results are consistent with the findings of other investigators (Abrams et al., 1991; Berkowitz et al., 1998; Wen et al., 1990). However, Kramer et al. (1995) did report an increased risk of PTB for women who had a poor gestational weight gain. The discrepancy between the findings of Kramer et al. (1995) and the findings of this study may be a result of methodological differences including: a) the use of different and more precise methods (i.e., total weight gain, rate of weight gain per week, or pattern of weight gain over each semester) for the assessment of the effect of maternal weight gain on birth outcome; and b) differentiation between the types of preterm delivery (spontaneous preterm delivery, prelabor rupture of membranes, medically indicated preterm delivery) and growth restriction (Abrams, Altamn, & Pickett, 2000; Berkowitz & Papiernik 1993; Carmichael & Abrams, 1997; Kramer, 1998; Schieve et al., 2000). In Kramer et al.'s study, poor gestational weight gain was operationalized as a weight gain of less than or equal to 0.27 kg/week. In comparison, in the current study inadequate gestational weight gain was operationalized as a weight gain of less than 0.5 kg/week *or* a weight loss from 26 to 36 weeks. Consequently, failure to find an association between inadequate weight gain and PTB may be due to the definition of what constitutes inadequate weight gain. In addition, Kramer included in his sample only women who had a spontaneous onset of labor prior to 37 weeks, and excluded women who had chronic illnesses, placenta previa, abruptio placenta, placenta previa, and severely growth retarded fetuses, whereas this study did not exclude these women.

In contrast to the null finding of the effect of poor gestational weight gain on PTB, women who had a poor gestational weight gain during pregnancy were at increased risk of delivering a LBW infant. The study findings are consistent with those reported by Chumnijaraki et al. (1992). A critical question to answer is why do disparate findings exist between the effects of low gestational weight gain and the outcomes of LBW and PTB? These differences may be a result of: a) a period of poor maternal weight gain that resulted in growth impairment; b) an interplay of other pregnancy-related factors with maternal weight gain that resulted in reduced fetal growth (Keen, 1993); and, c) poor gestational weight gain may have different influences depending on the specific etiological pathway for LBW (e.g., preterm delivery, inadequate fetal growth, or a combination of these processes).

Differential findings were also found for the effect of anemia on PTB and LBW. In this study, women with anemia had a 2-fold risk of delivering a LBW infant but they did not have an increased risk of delivering a preterm infant. The study findings for anemia and PTB were consistent with the results reported by Meis et al. (1995), Scholl et al. (1992, 1994), and Xiong et al. (2000), but were inconsistent with Scanlon et al. (2000) who reported an increased risk of PTB for women with anemia. Such differences may reflect how anemia was operationalized in the different studies. Scanlon et al. defined anemia in terms of hemoglobin. This value was adjusted for both gestational age and altitude, whereas in this study anemia was defined as a hemoglobin value of less than 100 g/L. Few studies have examined the effect of anemia on LBW. Comparisons with this study were not possible because investigators defined anemia in terms of hematocrit (Bondevik, Lie, Ulstein, & Kvale, 2001). The study findings regarding the effect of anemia on LBW but not on PTB may indicate that the biological processes of anemia (i.e., increase serum norepinephrine concentrations and elevated corticotrophin releasing hormone) influenced the growth of the fetus rather than initiating preterm labor, which would result in the delivery of a preterm infant (Allen, 2000).

Maternal Health and Pregnancy Complications

Several maternal medical problems have been linked to PTB and LBW (Coetzee & Levitt, 2000; Rudge, Calderon, Ramos, Abbade, & Rugolo, 2000; Samadi & Mayberry, 1998; Tan & Thomson, 2000). Several authors, however, suggest that the evidence for these associations must be interpreted with caution because there is also the possibility that the observed associations between these factors and PTB and LBW may actually be due to early medical intervention rather than on gestational duration and birth weight per se (Berkowitz & Papiernik, 1993; Tan & Thomson, 2000).

In this study, women with pre-existing chronic diseases (i.e., maternal chronic hypertension, renal disease, diabetes mellitus) had an increased risk for PTB. Similar findings have been reported in other studies (Feig & Palda, 2002; Meis et al., 1998; Samadi & Mayberry, 1998; Ray et al., 2001). The risk of PTB among women with chronic hypertension was 1.68. Other studies reported similar risk estimates ranging from 1.6 (Samedi & Mayberry, 1998) to 4.0 (Meis et al., 1998). Although women with renal disease had a 4-fold risk of PTB, it is important to note that the wide confidence interval suggests a lack of precision regarding this risk estimate.

Women with chronic hypertension were at increased risk of delivering a LBW infant. This impact of chronic hypertension on birth weight is also supported in the research literature (Ananth, Peedicayil, & Savitz, 1995; Velentgas, Benga-De, & Williams, 1994). It is not surprising that women with diabetes in the study were not an increased risk for delivering a LBW infant as infants of diabetic mothers are often macrosomic or large for gestational age (Coetzee & Levitt, 2000; Rudge, Calderon, Ramos, Abbade, & Rugolo, 2000).

In this study, other medical conditions such as asthma, Crohn's disease, and ulcerative colitis were aggregated into one category with the label acute medical problems. As such the separate effects of these medical problems on birth weight and gestational age were not estimated. Women with one or more acute medical problems had a 3-fold risk of PTB. Researchers have found a relationship between PTB and asthma (ORs ranged from 1.78 to 2.37) (Liu et al., 2001; Sorensen et al., 2003; Wen, Demissie, & Liu, 2001) and between PTB and ulcerative colitis (OR=3.4; CI: 1.8-6.4) (Norgard et al., 2000). Joint examination of the LBW analysis and preterm analysis suggests that the effect of acute medical problems on LBW was through its impact on gestational age.

In this study, the pregnancy complications of gestational bleeding, placenta previa, gestational hypertension, and gestational hypertension with proteinuria were associated with PTB. This is congruent with the findings of Lang et al. (1996), Meis et al. (1995), and Yang and Savitz (2001), who have determined the effect of one or more pregnancy complications on PTB after adjusting for other risk factors such as maternal age, previous obstetrical history, smoking, and education. Several studies have examined the association between pregnancy complications on adverse outcomes such as PTB. PTB risk estimates range from 5.5 to 11.0 for placenta abruption (Ananth et al., 1999; Lang et al., 1996), 2.7 to 4.4 for trimester-specific bleeding (Lang et al., 1996;Yang &

Savitz, 2001), and 2.4 to 5.0 for pre-eclampsia or toxemia (Lang et al., 1996, Clausson et al., 1998). In this study, with the exception of placenta previa, all pregnancy complications were significantly associated with LBW. In interpreting these findings, it is important to recognize that a) these pregnancy complications can lead to early delivery through medical intervention; and b) the birth outcome LBW is a function of an infant being born too soon.

The multivariate analyses in this study demonstrated that pregnancy complications had a significant effect on both gestational age and on birth weight. Moreover, the impact of pregnancy complications on PTB and LBW was greater for older women. These adverse outcomes may have been the result of early intervention due to the mother's or infant's health. As Kramer (1987) and other researchers (Anath et al., 1999; Lang et al., 1996; Sheiner, Shoham-Vardi, Hallak, Hershkowitz, Katz, & Mazor, 2001) have noted, it is controversial as to whether pregnancy complications and medical diseases (i.e., hypertension, diabetes mellitus) should be included in etiologic studies of LBW because these conditions are considered to be intermediate outcomes of pregnancy. Pregnancy complications were included in the current study to look at the direct effects of complications and to see if other factors are operating indirectly through these complications. Regardless of the existing methodological debate, the study findings draw attention to the importance of preconceptual counselling and sound prenatal care that informs women about risk factors that can impact pregnancy complications and pre-existing diseases and about their management options.

Socio-economic Status

In this study, socio-economic status (as measured by aggregate level data) did not have a direct effect on gestational age or birth weight. However, SES did have an indirect effect through its influence on other risk factors. The study findings suggest that living in a disadvantaged socio-economic environment influences the adequacy of prenatal care, maternal pre-pregnancy weight, medical problems both prior to and during pregnancy, and lifestyle behaviors such as street drug use. In the LBW model, SES exerted its effect through inadequate prenatal care, low-pre-pregnancy weight, and the use of street drugs. In the PTB model, SES operates through pre-existing diseases (other medical diseases), gestational diabetes, inadequate prenatal care, and gestational hypertension for older women.

The study findings differ from those of other investigators who have examined aggregate measures of SES and birth outcomes. Significant findings have been reported between census-based socio-economic factors and LBW (Crosse et al., 1997; O'Campo et al., 1997) and preterm delivery (Kaufman, Dole, Savitz, & Hering, 2003). However, these researchers did not examine simultaneously risk factors that were included in this study (e.g., smoking, street drug use, medical problems). It is therefore plausible that if these risk factors had been included in these analyses, then the area-based measures may not have demonstrated a significant direct effect. It is also possible that study differences are a result of the different area-based measures used in different studies.

In this study, the null finding of the effect of SES on PTB and LBW may be due to methodological problems in the measurement of SES and the inability to include all women in the study due to the unavailability of geographical PHSA data. Each measure of SES was limited to 17 data points (i.e., one for each PHSA) rather than 26,265 unique SES data points (i.e., one for each woman). This reduction limits the heterogeneity of SES within each PHSA and the ability to adequately assess if a relationship exists. Furthermore, each PHSA includes numerous census tracts; thus, the PHSA measure may not be an adequate proxy of the mother's immediate neighbourhood environment. Consequently, the effect of the PHSAs may have been lost by not having a more refined measure that captures the characteristics of the woman's neighbourhood (Pickett & Pearl, 2000). It is also possible that the five SES variables used to characterize the PHSA may not have adequately captured the differences across neighbourhoods or the pathways by which socio-economic environment translates into poor birth outcomes (Pickett & Pearl, 2001).

There is a growing body of literature suggesting that area-level social and economic disadvantage influences smoking behavior (Tseng, Yeatts, Millikan, & Newman, 2001; Stead, MacAskill, MacKintosh, Reece, & Eadie, 2001), street drug use

(Schroeder, Latkin, Hoover, Curry, Knowlton, & Celentano, 2001), and prevalence of medical problems (Green, Hoppa, Young, & Blanchard, 2003). Therefore, it is important to consider how socio-economic disadvantage may contribute to adverse birth outcomes related to a woman's health seeking behavior (e.g., prenatal care utilization), lifestyle factors (e.g., street drug use), and overall health status (e.g., pregnancy complications, medical problems). In this study, SES influenced birth outcomes through these factors.

The mapping process provided information about the geographical distribution and prevalence of these and other risk factors within the PHSAs. The maps revealed that women who resided in socially disadvantaged environments (PHSAs) did not necessarily have an increased prevalence of *all* risk factors that were predictive for PTB and LBW. However, Central, the most disadvantaged PHSA, did have PTB and LBW rates that were significantly higher than those of CHA. As well, Central had higher prevalences for many of the study risk factors. These maps provide a baseline for examination of the risk factor trends and can be used by providers and program planners to develop programs that target these factors within specific PHSAs.

There were some differences between the General PTB and LBW models and the SES PTB and LBW models. Comparison of the overall pattern of risk factors and the change in the magnitude of the odds ratios and confidence intervals, suggests that the differences in risk estimates were very small. It is important to point out that 1,387 women in the study (i.e, those on PHSA #18) were not included in the SES models because these women did not have socio-economic data. Thus, differences noted between the General and SES models for PTB and LBW may partially be a reflection of the exclusion of these women.

Collectively, the results of the models and the maps do encourage further exploration of why differences in risk factors exist and of how characteristics of PHSAs that are geographically defined operate at an aggregate level to influence behaviors and outcomes. It has been proposed by a number of investigators that the community or neighbourhood can affect health outcomes by influencing the stressors to which individuals are exposed, the resources available to deal with these stressors, and the individual's values, beliefs, attitudes, and social interactions (Diez et al., 1997; Reis, Mills-Thomas, Robinson, & Anderson, 1992). Although the study data cannot validate the existence of such mechanisms, it is plausible that these environmental factors may influence health promoting behaviors of women (i.e., smoking, street drug use), as well as their health status (i.e., pre-existing diseases) and the use of health services (i.e., prenatal care).

In summary, the analyses have demonstrated that risk factors have direct and/or indirect effects on PTB and LBW. It is important not to ignore the indirect effects of risk factors because they offer opportunities for intervention and they help us to understand more about the pathways leading to poor birth outcomes. In this discussion, several explanations have been offered as to why some study findings were inconsistent with the results of other studies. It is also possible that the disparate findings exist because many studies only report factors that have a direct effect. Some of the non-significant factors found in other studies may influence birth weight and/or gestational age indirectly through other factors.

Strengths and Limitations of the Study

Before discussing the implications of the study results, it is necessary to consider the methodological strengths and limitations of the study. These methodological considerations are important because they affect the validity of the study findings.

There were several study strengths. First, the study was a retrospective, population-based cohort study that included all births in CHA with the exception of women not delivering in a hospital. Therefore, the problem of selection bias was minimized. Nevertheless, there were birth records that could not be linked and were not included in the analyses. Comparison of specific maternal risk factors and infant outcomes of the study sample and the unlinked records did reveal significant differences. However, it is thought that any bias in the estimation procedure as a result of selection bias would be minimal. Furthermore, any resulting bias would lead to an underestimation of the computed odds ratios. For risk factors bordering on significance, Type II errors are possible.

Another strength of the study was the ability to consider concurrently a large number of maternal characteristics as potential predictors of both PTB and LBW. This analysis was facilitated by the relatively large sample size and the use of extensive computerized perinatal and vital statistics databases. These factors permitted control of a number of potential confounders. Other researchers have used limited data sources (e.g., birth certificates), and consequently, there were limitations in the number of variables that could be examined, including relevant confounders.

Reporting bias was minimized in the study. The majority of the data used in the analysis were collected prospectively before the birth outcome was known. Although the PNOB form was completed within the first 24 hours of the birth, the assessment of the potential risk factors would have been completed prior to the birth outcome, and thus reporting bias would be minimized.

Finally, the potential for misclassification of the preterm birth was minimized. Ultrasound fetometry is a routine part of obstetrical care in Alberta. Therefore, the potential misclassification of prematurity in those cases where the gestational age was not confirmed by ultrasound fetometry was likely negligible.

There were also several study limitations. The results of recent research suggest that there are different etiologic pathways to LBW (Lang et al., 1996, Michielutte et al., 1992). One limitation of this study was the inability to differentiate clearly between preterm delivery and growth restriction by operationalizing LBW into three categories (i.e., preterm appropriate for gestational age infants (P-AGA), preterm small for gestational age infants (P-SGA) and term small for gestational age infants (T-SGA). Although the categorization of P-AGA, P-SGA and T-SGA was not possible, the inclusion of gestational age in the LBW model, and the consequent joint examination of the LBW and PTB analyses, did provide insight into both the direct and indirect pathways leading to LBW. A further limitation was the inability to differentiate between spontaneous PTB (i.e., spontaneous labor, premature rupture of membrane) and PTBs that occurred as a result of early medical intervention (i.e., medically induced PTBs).

As noted in the methods section, there were also three variables (i.e., street drugs, prenatal visits, prenatal classes) with substantial missing data. As a result, the technique proposed by Cohen and Cohen (1984) for creating a new variable for each variable for which data is missing was used to avoid losing all of the data from subjects with missing data. This technique has been reported as being biased (Schopflocher, D. personal communication, April 2002). However, we cannot with certainty determine if this misclassification was nondifferential (resulting in an underestimate of the odds ratio) or differential (resulting in either underestimation or overestimation of the true odds ratio).

It is also possible that information bias may exist in area-based socio-economic measures utilized in the study. Desmissie et al. (2000) suggest that the misclassification associated with area-based measures is likely to be nondifferential resulting in an attenuated exposure outcome regression estimate, which can result in failure of the exposure to meet statistical significance. In this study the SES factors did not reach statistical significance. It was proposed that this null finding may result from the reduced heterogeneity of the SES within the PHSAs. The SES area-based measures were an aggregation of the census tracts for each of the PHSA. This aggregation limits the ability to detect small differences in the study population (Wilkins, Sherman, & Best, 1991).

Confounding is a third type of bias that threatens the internal validity of this study. Residual confounding and confounding at the contextual level have been identified as limitations in using area-based measures (Diez- Roux et al., 1997; Krieger et al., 1997). Residual confounding may occur when individual level socio-economic variables are controlled for in the analysis. In this study individual level socio-economic data were not available; thus, residual confounding by individual level SES data was not possible in the study. Confounding may also occur at the "contextual" or neighbourhood level (Diez et al., 1997). Individuals may form a part of a variety of contexts, many of which may overlap. For example, if neighbourhoods are segregated on the basis of women's relation to work, persons within that neighbourhood may share similar work environments and characteristics of these environments may be related to poor pregnancy outcomes. There were no study data to indicate that women share the same work environment. The study data only indicated that the women lived in the same geographical environment. It is plausible that there are social processes within the PHSA that can affect pregnancy outcome, thus, it is possible that contextual confounding may have existed in this study.

The study population was limited to those women who were residents of CHA and delivered a liveborn singleton infant with no major anomalies. Therefore, the results of the study can only be generalized to women who delivered a liveborn singleton infant with no major anomalies. Furthermore, it is not possible to say that any of the predictors identified in the study "caused" either PTB or LBW. Rather, it can only be stated that the factors were associated with the study birth outcomes (i.e., PTB, LBW).

Implications for Nursing Practice, Program Development, and Policy

Prevention of PTB and LBW is a major challenge because these birth outcomes are not restricted to an exclusive group of women with certain characteristics. Moreover, there are multiple risk factors and there remains uncertainty about the causal mechanisms (Best Resource Centre, 1998). Because of this complexity, prevention cannot be limited to one simple intervention provided by one specific health care provider. Instead, a community-wide (population health) approach that combines interventions directed at individuals with interventions to create supportive environments and healthy public policies is needed (Hamilton & Bhatti, 1996; Moutquin, 1998; Stewart, 1998b; Stewart & Nimrod, 1993). A population health approach encourages the community to focus on the underlying determinants that can influence individual health and behavior. It requires that individuals, families, health care providers, and various community sectors (i.e., education, health care, social services) work together to influence the health of childbearing families. A community-wide approach is necessary for several reasons: a) reduction within a high risk subgroup (i.e., younger women, older women, low-income women) will not have a major effect on the LBW population as a whole because the majority of births occur among women outside of these subgroups; b) approximately 70% of LBW infants are preterm and only 15-30% of PTBs occur in women known to be at increased risk (Moutquin et al., 1998); and c) focusing on only one risk factor will have little overall effect on the LBW or PTB rate.

What role can nurses play in the prevention of PTB and LBW and in the reduction of the morbidity associated with these adverse birth outcomes? The study findings suggest several implications for nursing interventions: a) increasing utilization of prenatal care services; b) assessment of risks related to lifestyle behaviors, medical problems during pregnancy, and pregnancy complications; and, c) providing health promotion and preventive counselling to women regarding smoking cessation, and early identification and treatment of medical problems and pregnancy complications.

The study findings indicate that women with inadequate prenatal care (i.e., no prenatal classes, less than 8 prenatal visits) have an increased risk of having a preterm or LBW infant. Moreover, younger women and women in socially disadvantaged environments appear to be at greater risk for inadequate prenatal care. It is not unreasonable, therefore, to suggest that greater efforts need to be made to ensure that prenatal care services are financially and geographically accessible and are culturally and socially relevant to the particular needs of the different groups of women. While nurses can play a vital role in preconceptual care and in prenatal care of all childbearing families within their communities, it is also apparent that more innovative approaches to increase access and utilization of prenatal care services are needed for subgroups of women at greater risk.

Despite universal coverage of prenatal medical care in Alberta, the study findings suggest that there are other barriers that must be identified in an effort to develop more effective programs and services. Barriers that have been cited as hindering prenatal care utilization by low-income women include service delivery dimensions (i.e., waiting times, appointment difficulties, dissatisfaction with care, and poor relationships with providers); intrapersonal factors (i.e, excessive stress, demands on time; lack of perceived need); situational factors (i.e., transportation and child care); community factors (i.e., social norms); and interpersonal factors (i.e., lack of support from social networks) (Aved, Irwin, & Cummings, 1993; Fischler & Harvey, 1995; Loveland-Cook, Selig, Wedge, Gohn-Baube, 1999; Stout, 1997; Sword, 1997). The existing structure and delivery of prenatal care services and prenatal classes within Alberta may not be designed to address adequately the critical concerns related to social disadvantage. Stewart et al. (1996) suggest that more accessible and appropriate care may be provided by outreach programs and multidisciplinary teams sensitive to the needs of women who are marginalized in society because of low income, low education, being a teen, or being single.

There are several examples of prenatal programs that address barriers to prenatal care for women at risk for adverse birth outcomes. One highly successful program that offered targeted support to high-risk inner-city low-income Hispanic women and their families in Houston, Texas was the de Madres à Madres Program (Mc Farlane & Fehir, 1994). The premise of this program was that culturally relevant support by indigenous volunteer mothers in conjunction with community resource information would enable pregnant women to overcome the barriers preventing early prenatal care and thus "open the door" to health care and social services for themselves and their families. This program was successful in increasing access to and utilization of health and other services for pregnant women, their families, and the community, as well as achieving healthy infant outcomes. For example, over a four-year period, not one LBW infant was born to a woman followed by a volunteer mother of de Madres à Madres. Successful community-based maternal and infant health programs have also been developed for pregnant adolescents and mothers (Flynn, 1999; Lapieere, Perrault, & Goulet, 1995). Central elements of these programs were health counselling, peer support, home visitation or school based clinic visits by paraprofessionals who are indigenous to the community, and partnership with community health nurses and other providers in the provision of health and other services. These programs demonstrate how nurses can work with communities to develop targeted programs, and the importance of developing programs that are socially and culturally appropropriate to the needs of the community and its residents.

Community health nurses, midwives, and nurse practitioners focus on the broader social, economic, and political issues that shape a woman's life. Consequently, they are well positioned to work with women, other service providers, and community leaders to develop and implement a holistic model of prenatal care that addresses the specific needs of childbearing women, especially, younger women and women who live in socially disadvantaged environments.

Although this study could not determine the type of assessment or advice that women received during their prenatal care visits or prenatal classes, both Canadian and American studies suggest that not all women are being counselled about general risk reduction strategies or about the signs and symptoms of pregnancy complications such as premature rupture of membranes, gestational hypertension, or preterm labor (Davies et al., 1998; Petersen et al., 2001). The increased smoking prevalence across many PHSAs of CHA, the associated risks of LBW for women who smoke, the negative effect of street drug use on LBW, and the associated risk of PTB and LBW for women with pregnancy complications and medical conditions, all confirm the importance of consistent prenatal assessment and counselling. Maloni (2000) suggests that providers have a dual responsibility of educating the community regarding risk factors, treatment of risk conditions such as pregnancy complications, and working with the community to reduce the barriers that prevent women from endorsing health promotion and risk reduction strategies. Public transportation, supermarkets, television, and community publications may provide several venues for delivering such educational messages to pregnant women.

In addition to enhancing access to relevant prenatal care and education, nurses can also play a vital role in decreasing risk behaviors that negatively influence birth outcomes. Smoking remains the single most modifiable risk factor for LBW (Adams & Melvin, 1998; Adams & Young, 1999), with population attributable risk percentages ranging from 17% to 34% for LBW (Cnattingius & Haglund, 1997; Lightwood, Phibbs, & Glantz, 1999). It is this evidence that makes smoking an important public health issue (Lowe & Wakefield, 1998; Lumley et al., 2002; Maloni, 2000; Messecar, 2001). In this study, there are several indications of the importance of this issue -- a high rate of smoking among pregnant women within the PHSAs of Capital Health, a 2-fold risk of LBW for women who smoke, and the apparent effect of smoking on PTB through gestational bleeding. All of these factors support smoking prevention and smoking cessation programs and policies as important intervention strategies within CHA.

Both the study data and the existing economic studies about the escalating maternal and neonatal costs associated with smoking during pregnancy (Adams & Melvin, 1998; Adams & Young, 1999; Lightwood et al., 1999) convincingly support the argument that it is crucial to undertake strategies in the population at large, including women of childbearing age, their partners, and the communities in which they live. Lightwood et al. (1999), in a California study of economic benefits of smoking cessation, reported that an annual drop of one percentage point in smoking prevalence would prevent 1,300 LBW births, thus saving 21 million dollars (in 1995 dollars) in direct medical costs in the first year of the program and 572 million dollars (in 1995 dollars) in direct medical costs over seven years. Specific neonatal costs attributable to maternal smoking during pregnancy have also been estimated. Analysis has shown that maternal smoking increases the admission to NICU by 20%, and increases the length of stay by 1.1%. The smoking attributable neonatal costs in the US represent almost $367 million in 1996 dollars (Adams, Miller, Ernst, Nishimura, Melvin, & Meritt, 2002).

These American cost estimates should encourage CHA planners and policy makers to evaluate short-term and long-term maternal, infant, and childhood smoking-related costs, to examine existing smoking prevention and cessation initiatives within PHSAs with high smoking prevalences, and to support the development, implementation, and evaluation of both current and future smoking cessation initiatives. While it is ideal to have smoking interventions directed at a regional level, resource limitations may mean deciding to first target PHSAs with smoking prevalences that are unacceptably high (i.e., above the national smoking prevalence target of 20%) (Steering Committee of the National Strategy to Reduce Tobacco Use in Canada and the Advisory Committee on Population Health, 1999).

The evidence to support smoking cessation programs during pregnancy is growing (Lumley, Oliver, & Waters, 2002). Lumley et al. (2002), in a review of 34 clinical trials for the Cochrane Collaboration, revealed that smoking cessation programs during pregnancy are effective in reducing smoking, LBW, and PTB. Interventions commonly used in these programs were: counselling to quit or reduce smoking during pregnancy; providing information on the risks of smoking to the fetus and infant, and on the benefits of quitting; and teaching cognitive behavioral strategies for quitting smoking. There was variation in the intensity of the intervention and the extent of reinforcement and support throughout pregnancy across the different trials.

Attention to smoking behaviour, together with assistance and support to reduce or stop smoking, needs to be as routine a part of antenatal care as the measurement of blood pressure (Lumley et al., 2002). As a component of preventive and health promotion counselling with each prenatal care visit, providers must assess the fifth vital sign "tobacco use" (Fiore, Jorenby, Schensky, Smith, Bauer, & Baker, 1995). As part of this assessment, practitioners need to determine why a woman smokes, what contextual elements in a woman's life reinforce smoking, and how motivated and ready the woman is to change her smoking behavior (Stead et al., 2001; Secker-Walker, Solomon, Flynn, Skelly, & Mead, 1998; Wakschlag et al., 2003; Woodby, Windsor, Snyder, Kohler, & DiClemente, 1999). Haslam, Draper, and Goyder (1997) and DiClemente, Dolan-Mullen, and Windsor (2000) concur that understanding the obstacles and pathways for smoking cessation are critical in guiding health care providers and in developing and implementing effective programs.

Several studies have provided insight into why pregnant women smoke. Floyd et al. (1993) suggest that pregnant women continue to smoke for many of the same reasons that they smoked prior to the pregnancy (e.g., stress, lack of knowledge, do not perceive benefits of quitting, no support to quit smoking). Women who live in disadvantaged environments are more likely than other women to smoke during pregnancy (Steering Committee of the National Strategy to Reduce Tobacco Use in Canada and the Advisory Committee on Population Health, 1999; Wakschlag et al., 2003). Numerous investigators have documented the reasons for continued smoking and the barriers to smoking cessation among low-income women and low-income women who are pregnant (Graham, 1994; Haslam, 1999; Najman, Lanyon, Andersen, Williams, Bor, & Callaghan, 1998; Stead, et al, 2001; Stewart, Gillis, Brosky, Johnston, Kirkland, Leigh, Persuad, Rootman, Jackson, & Pawliw-Fry, 1996; Wakefield & Jones, 1998; Wakschlag et al., 2003). A common theme in these studies is that smoking provides women with a means for coping with the stress of material inadequacies, isolation, loneliness, powerlessness, and limited social opportunities. Major smoking cessation barriers identified by women include the lack of support from partners, friends, and family; the lack of meaningful support and assistance from health care and other service providers; and the presence of pro-smoking community norms. The development of smoking cessation programs within the disadvantaged PHSAs in this study (e.g., Central, North Central, and West Central) means that program developers will need to address the stressors faced by these women; the powerful influence of partners, friends, family, and community norms; and the resources available in the community.

One model that has been proposed to guide smoking cessation intervention is Prochaska and DiClemente's (1983) stages of change model. This model proposes that smokers move through different stages of motivation and psychological readiness to change in relation to smoking cessation. These stages include: pre-contemplation (not considering quitting), contemplation (thinking about quitting), preparation (making plans to quit), action (individual has changed behavior and quits for a period of time), and maintenance (has quit for an extended period and works to prevent relapse). Health professionals using this model could determine the relevant stage of change of the pregnant smoker, monitor movement through stages, and tailor antenatal smoking cessation material and information to the level of intention to quit (Haslam, 1999; Stead et al., 2001).

This model has been used to determine if a pregnant women's stage of change is associated with her assessment of the health risks related to smoking (Haslam & Draper, 2000) and to examine the motivation of pregnant women who quit smoking or continue to smoke (De Vries & Backbier, 1994). The combined results of these studies suggest that acceptance of health risks associated with maternal smoking varied according to the stage of change (Haslam & Draper, 2000) and attitudes towards cessation varied according to stage of change. The results of these studies lend support to Prochaska et al.'s (1994) argument that smoking cessation efforts need to 'place' the recipients in terms of their stage of change and the need to target information accordingly.

Clinicians who provide health care to women have an important role in reducing the burden of smoking among women (Ebrahim, Floyd, Merritt, Decoufle, & Holtzman, 2000; Kirkland, Dodds & Brosky, 2000). Beyond counselling to individuals, however, it is also essential that collective actions be taken to alter those conditions or events that support smoking behavior. Nurses can advocate with others for policies such as prohibition of smoking in all public places; tobacco control strategies for pregnant women, young women, and low-income women; and for workplace and community smoking prevention and cessation programs.

Although the importance of smoking cessation during pregnancy cannot be overstated, smoking prevention strategies must target youths and their families throughout the community to provide a consistent message to young people who are the future childbearing population. More specifically, efforts must be directed at preconceptional counselling and general health counselling across the reproductive lifespan of women and men. Ebrahim et al. (2000) suggest that long-term reduction in tobacco exposure during pregnancy can be achieved by encouraging teenage girls and young women not to start smoking.

The mapping of risk factor prevalences in the different PHSAs indicates that there are still a number of women within CHA that are not receiving regular prenatal visits or attending prenatal classes. Consequently, these women may miss opportunities for assessment, health promotion and prevention counselling, as well as referrals to valuable programs and services. Davies et al (1998), in a population survey of health care professionals in Ontario, Canada, reported that a considerable proportion of women are not receiving lifestyle counselling or counselling regarding the signs and symptoms of pregnancy complications and their management. It may be argued that within Alberta, the current shortage of obstetricians and family physicians, the current fee-for-service environment, and the delays in acknowledging midwifery practice within existing health reimbursement schedules are creating a system where there is insufficient time for practitioners to provide health promotion activities and important primary and secondary prevention interventions to all pregnant women. It is proposed that creative models of prenatal care services that include a multidisciplinary team, paraprofessionals, outreach services, and partnerships with other community agencies may better support the specific needs of both 'at risk' mothers and other childbearing families within CHA.

Implications for Future Research

This study utilized existing administrative databases to describe the distribution of risk factors and birth outcomes across the PHSAs of CHA and to determine the association between maternal and newborn characteristics and adverse outcomes, that is, PTB and LBW. The study findings indicate that while there were PHSAs that had similar risk factor prevalences, there were also differences in risk factor prevalences across the PHSAs. Furthermore, the risk factor pattern did not indicate that those PHSAs that had LBW and PTB rates higher than CHA also had higher prevalences of all risk factors that were predictive of LBW or PTB. An important component of future surveillance research within CHA will be to monitor both risk factor and birth outcome trends. Future studies should also consider the heterogeneity of PTB (i.e., different etiological pathways by clinical presentation-- preterm labor, premature rupture of membranes, medically induced PTB) and the direct and indirect influences of previously identified risk factors on these outcomes. Risk factors may have a different impact depending on the pathway leading to preterm delivery and/or fetal growth restriction.

To improve existing perinatal health surveillance research both within CHA and Alberta, database managers and practitioners need to continue to work together to ensure that regional and provincial databases provide a comprehensive provincial perinatal surveillance system. Existing databases, such as the Northern and Central Alberta Perinatal Audit Database and the Provincial Notice of Live Birth or Stillbirth are not sufficiently comprehensive for monitoring the various components of perinatal health. To be able to develop a more complete etiologic model and extend our understanding of the direct and indirect determinants that influence birth weight and/or gestational age, it is important that the provincial database include biological markers and other important risk factors that have been identified in the literature (e.g., stress, infection). Concerted efforts must also be made to expand existing databases to include factors such as continuous measures of pre-pregnancy height and weight, weight gain per week, individual measures of socio-economic status (i.e., income, education), race, content and timing of prenatal care, use of health and social services programs, and use of artificial reproductive technology (e.g., IVF). To enhance examination of geographical and socio-economic variations in perinatal health, attention must also be given to completion of postal codes and easier access and utilization of census data. Inclusion of data about program referral and preventive counselling may be useful in determining the contribution of these efforts to changing the prevalence of modifiable risk factors (i.e., smoking, street drug use) and birth outcomes.

To compare risk factors and birth outcome trends across the country, it will be essential that regional and provincial surveillance systems include indicators that are parallel to the Canadian Perinatal Surveillance System (Dzakpasu, 1998). While it is important to have an inclusive database, its effectiveness is dependent on the conscientious and consistent completion of all forms that support the databases.

Information derived from such a comprehensive surveillance system would be relevant to policy makers in their efforts to monitor provincial trends in perinatal health status indicators, to calculate the occurrence of adverse pregnancy outcomes, to identify the contributing risk factors, and to describe and explain geographic differences in perinatal outcomes within and across RHAs. This information could then be used by policy makers, health planners, and administrators in RHAs for planning and resource allocation, for the development and implementation of population-based prevention programs, and for relevant health promotion strategies designed to improve the health of Alberta women and their infants.

The evidence from the PHSA maps and the risk modeling indicates a need to target programs in PHSAs with high risk prevalences (e.g., smoking, prenatal care utilization). As providers and policy makers attempt to meet the LBW rate target, it will be important to evaluate the effects of existing and new smoking prevention and cessation program initiatives on smoking behavior and pregnancy outcomes. An important component of this research could be the development and evaluation of stage-dependent antenatal smoking cessation material and identification of individual and community factors that enhance or hinder smoking reduction or smoking cessation efforts.

Future research could also examine the effect of risk factors for which there continues to be inconclusive evidence about their impact (e.g., stress, environmental toxic exposures, and prenatal care). There is a need for more research to consider what constitutes adequate prenatal care (Kogan et al., 1994) and how to measure the effectiveness of prenatal care on maternal and infant outcomes (Alexander & Kotelchuck, 1996). To examine the impact of prenatal care on birth outcomes, researchers indicate that it is necessary to consider the quality and content of prenatal care, the timing of the initiation of care and number of visits (to adjust for high risk pregnancies and early delivery for medical reasons), as well as controlling for potential confounders (Alexander & Kotelchuk, 2001; Delvaux, Beukens, Godkin, & Bousten, 2001).

More specifically, prenatal care research should examine what preventive counselling was received by women (from the perspectives of both provider and the woman), the sources of this information (written materials, counselling by provider, childbirth education), whether the information was used with a resulting change in behavior, what resources and barriers either facilitated or inhibited change, what interventions for mother and infant were used, and what were the outcomes. Similarly, it is important to determine among women who do not access prenatal care or have limited prenatal care: a) the reasons for not accessing care; b) what health behaviors women have changed to promote a healthy birth outcome; c) what have been the barriers in fostering a change in behaviors; d) what helped women change their behaviors; and e) what are the outcomes for neonates whose mothers did not receive care or had limited prenatal care. It would also be essential to evaluate how changes in the health system can influence the use and outcome of prenatal care.

In addition, there is a need in prenatal care research to examine closely whether the birth outcome measures of PTB and LBW are the most appropriate measures to examine the effectiveness of prenatal care. Current interventions (i.e., corticosteroids, tocolytics) are leading to improved survival rates of infants (preterm and LBW infants) who previously would have been considered nonviable (i.e., less than 24 weeks) (Gregory & Davidson, 1999; Helfand Zimmer-Gembeck, 1997; & Kogan, Martin, Alexander, Kotelchuk, Ventura, & Frigoletto, 1998). Thus, using these birth outcomes as the primary measures of the effectiveness of prenatal care may not be appropriate because these measures do not reflect the positive effects of current interventions. Prenatal care outcome measures such as adoption of health promoting and prevention behaviors and satisfaction with care received may be more promising outcomes (Alexander and Kotelchuk, 2001; Bell-Woodward & Edouard, 1992; Fischler & Harvey, 1995).

Although socio-economic status was not a predictor of LBW or PTB in this study, the data do appear to suggest that the woman's socio-economic environment may play a role in her health or her infant's health. One of the most robust findings in perinatal epidemiological research concerns the large socio-economic disparities in key pregnancy outcomes such as perinatal mortality, LBW, and PTB (Kramer, Goulet, Lydon, Sequin, McNamara, & Das, 2001; Luginaah et al., 1999; Parker et al., 1994; Pearl et al., 2001; Rolett & Keily, 2000). Although many explanations have been proposed, there are a few studies that have examined the role of community and other social, economic, and structural factors as potential causal pathways that may explain these social disparities (O' Campo et al., 1997; Rauh et al., 2001). Preliminary research suggests that individual risk factors on birth outcomes are moderated by neighbourhood characteristics; and as well, neighbourhood characteristics have an effect independent of individual-level attributes (O'Campo, et al, 1997; Rauh et al., 2001). These findings and the results of the study suggest that there is a need to include both individual and aggregate measures of SES to determine if these measures differ from each other in terms of their impact on birth outcomes.

To gain a more comprehensive understanding of the role of SES, these measures must be analysed concurrently with a more comprehensive list of individual level risk factors. In addition to selected factors examined in this study, measures of stress and social support are factors that have been identified in the LBW literature as plausible mechanisms in the causal pathway between SES and birth outcome (Austin & Leader, 2000; Dejin-Karlsson, Hanson, Ostergren, Lindgren, Sjoberg, & Marsal, 2000; Dunkel-Schetter, 1998; Feldman, Dunkel-Schetter, Sandman, & Wadwha, 2000; Hoffman & Hatch, 1996; Kramer et al., 2001; Mackey, Williams, & Tiller, 2000; Paarlberg, Vingerhoets, Passchier, Dekker, Heinen, & van Geijn, 1999; Sheehan, 1998; Wadwha, Culhane, Raugh, Barve, Hogan, & Sandman, 2001).

Investigating the relationship between geographic areas and birth outcomes requires using several different research designs. Quantitative designs need to include data on both the women and the areas in which they live. Furthermore, the studies need to include adequate numbers of neighbourhoods and individuals within the neighbourhoods to permit examination of within and between neighbourhood variability in the outcomes and in the factors associated with them (Diez Roux, 2001). An important aspect of these designs will be to determine the size and definition of the relevant geographical area, identify potential data sources, and hypothesize on the processes through which neighbourhood and individual factors may jointly and independently influence outcomes. However, qualitative study designs may also be helpful in understanding how area and individual characteristics operate in influencing birth outcomes. An essential component of investigating neighbourhood effects on the health of pregnant women and their infants are studies that evaluate the effect of policies or interventions targeted at improving neighbourhoods.

Conclusion

The prevention of PTB and LBW is a public health priority. To date prevention programs directed toward women at high risk, or efforts focused on institution-specific and high technology medical approaches, have been ineffective in reducing PTB or LBW rates (Alexander, 1998; Moutquin et al., 1996). Present thinking is that a population health approach that considers the "living and working environments that affect people's health, the conditions that enable and support people in making health choices, and the services that promote and maintain health" (Federal, Provincial, and Territorial Advisory Committee on Population Health, 1994, p.9) will better influence pregnancy outcomes for the population as a whole.

The results of the study identified risk factors that had a direct and/or an indirect effect on birth weight and gestational duration. There were risk factors that were common to both PTB and LBW and unique to these birth outcomes. Although some of the risk factors were non-modifable, several modifiable risk factors such as prenatal care and lifestyle behaviors were identified. The study also identified that the distribution of risk factors and birth outcomes varied across geographic areas of CHA.

These findings should assist nurses and other providers to develop more effective primary and secondary prevention interventions. The results of the study suggest that innovative approaches are required to increase utilization of prenatal care services and that prevention strategies need to be directed at modifiable risk factors, especially smoking. As well, secondary prevention strategies need to be directed at early detection and treatment of medical and pregnancy complications.

Nurses have a rich history of providing preventive and population-based care (Butterfield, 1990; Keller, Strohschein, Lia-Hoagberg, & Schaffer, 1998; Kuss, Proulx-Girouard, Lovitt, Katz, & Kennelly, 1990). Consequently, they are in a strategic position to endorse, implement, and to evaluate population-based approaches that utilize primary and secondary prevention strategies that focus on individual and area-based determinants of LBW and PTB. For example, knowledge of the direct and indirect effects of risk factors and the distribution of risk factor prevalences provides direction for counselling of pregnant mothers and their partners and for creating conditions and resources within the community that support the needs of pregnant families.

The array of factors influencing LBW and PTB suggest that these adverse birth outcomes are a community issue. Multiple stakeholders across numerous sectors must continue to put PTB and LBW on the political, educational, social, and health agendas of municipal, provincial, and national governments. Collectively, actions must be taken to fund the development and implementation of appropriate interventions that address the etiology of PTB and LBW.

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Appendices

Appendix A: Federal Census Data (1996) for Capital Health Region and Public Health Services Areas within the Capital Health Region

Federal Census Data (1996) for Capital Health Region and Public Health Services Areas within the Capital Health Region

Area	Demographic Characteristic	Attribute of Area
Capital Health	Single Parent Families	16%
	Less Than Grade 9 Education	7%
	Baccalaureate Degree or Higher	15%
	Median Income of Census Families	$49,500
	Aboriginal Population	3.7%
St Albert Area	Single Parent Families	11 %
	Less Than Grade 9 Education	2.0%
	Baccalaureate Degree or Higher	17%
	Median Income of Census Families	$ 62,000
	Aboriginal Population	2.0%
Castle Downs Area	Single Parent Families	17%
	Less Than Grade 9 Education	7%
	Baccalaureate Degree or Higher	8%
	Median Income of Census Families	48,000
	Aboriginal Population	2.0
West Central Area	Single Parent Families	20%
	Less Than Grade 9 Education	8%
	Baccalaureate Degree or Higher	16%
	Median Income of Census Families	$39,500
	Aboriginal Population	5.7%
Central Area	Single Parent Families	24%
	Less Than Grade 9 Education	16%
	Baccalaureate Degree or Higher	9%
	Median Income of Census Families	$30,000
	Aboriginal Population	9.8%
North Central Area	Single Parent Families	17%
	Less Than Grade 9 Education	13%
	Baccalaureate Degree or Higher	7%
	Median Income of Census Families	$41,000
	Aboriginal Population	5.1%

Federal Census Data (1996) for Capital Health Region
and Public Health Services Areas within the Capital Health Region

Area	Demographic Characteristic	Attribute of Area
North East Area	Single Parent Families	20%
	Less Than Grade 9 Education	9%
	Baccalaureate Degree or Higher	6%
	Median Income of Census Families	$43,000
	Aboriginal Population	6.3%
West (North) Area	Single Parent Families	18%
	Less Than Grade 9 Education	8%
	Baccalaureate Degree or Higher	13%
	Median Income of Census Families	$47,500
	Aboriginal Population	2.0%
West (South) Area	Single Parent Families	15%
	Less Than Grade 9 Education	3%
	Baccalaureate Degree or Higher	21%
	Median Income of Census Families	$58,000
	Aboriginal Population	1.8%
South West (West) Area	Single Parent Families	10%
	Less Than Grade 9 Education	3%
	Baccalaureate Degree or Higher	40%
	Median Income of Census Families	$67,500
	Aboriginal Population	1.1%
South West (East) Area	Single Parent Families	17%
	Less Than Grade 9 Education	5%
	Baccalaureate Degree or Higher	26%
	Median Income of Census Families	$47,500
	Aboriginal Population	2.3%

Federal Census Data (1996) for Capital Health Region and Public Health Services Areas within the Capital Health Region

Area	Demographic Characteristic	Attribute of Area
South Central Area	Single Parent Families	15%
	Less Than Grade 9 Education	8%
	Baccalaureate Degree or Higher	16%
	Median Income of Census Families	$47,000
	Aboriginal Population	2.6%
Millwoods Area	Single Parent Families	16%
	Less Than Grade 9 Education	6%
	Baccalaureate Degree or Higher	12%
	Median Income of Census Families	$47,500
	Aboriginal Population	2.7%
Sherwood Park Area	Single Parent Families	10%
	Less Than Grade 9 Education	2%
	Baccalaureate Degree or Higher	16%
	Median Income of Census Families	$67,000
	Aboriginal Population	1.2%
Strathcona County Area	Single Parent Families	6%
	Less Than Grade 9 Education	4%
	Baccalaureate Degree or Higher	12%
	Median Income of Census Families	$59,500
	Aboriginal Population	1.2%
Thorsby Area	Single Parent Families	8%
	Less Than Grade 9 Education	11%
	Baccalaureate Degree or Higher	5%
	Median Income of Census Families	$43,500
	Aboriginal Population	2.6%

Federal Census Data (1996) for Capital Health Region and Public Health Services Areas within the Capital Health Region

Area	Demographic Characteristic	Attribute of Area
Leduc Area	Single Parent Families	11%
	Less Than Grade 9 Education	2%
	Baccalaureate Degree or Higher	17%
	Median Income of Census Families	$62,000
	Aboriginal Population	2.0%
Beaumont Area	Single Parent Families	9%
	Less Than Grade 9 Education	5%
	Baccalaureate Degree or Higher	10%
	Median Income of Census Families	$56,500
	Aboriginal Population	2.3%

Appendix B: Assembled File: NCAPAPD and the Combined Alberta Vital Statistics and PNOB

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
Identification Information				
linked	N/A	linked	Linkage ID created for every database. Identifies rules for link	Defined by birth record
recnum	N/A	recnum	Birth record number	Defined by case
baby hospital	NCAPAPD	babhos	Baby's hospital number	Defined by case
vs_regno	AlbertaVital Stats/PNOB	vs_regno	Vital statistics registration number	Defined by case
deliver1	NCAPAPD	deltime	Delivery time	Defined by case
deliver2	NCAPAPD	bthr_pn	Delivery hour	Defined by case
brth_hr	Alberta Vital Statistics/ PNOB	bthr_ob	Birth hour	Midnight=00 Enter as on form
Linkage Information				
delivery	NCAPAPD	dob_pn	Baby's birth date	Defined by case
dob	Alberta Vital Statistics/ PNOB	dob_vs	Baby's birth date	Defined by case
mother_a	NCAPAPD	mage_pn	Mother's age at delivery	Defined by case
mother_a	Alberta Vital Statistics/ PNOB	mage_vs	Mother's age at delivery	Defined by case
living	NCAPAPD	lvg_pn	Previous live births: Excludes current	Defined by case
living	Alberta Vital Statistics/ PNOB	lvg_vs	Previous live births: Excludes current	Defined by case
gestatia	NCAPAPD	gest_pn	Gestational age of infant at birth (weeks)	Defined by case
gestatio	Alberta Vital Statistics/ PNOB	gest_vs	Gestational age of infant at birth (weeks)	Defined by case
sex	NCAPAPD	sex_pn	Baby's sex	F=female M=male O=not known
sex	Alberta Vital Statistics/ PNOB	sex_vs	Baby's sex	F=female M=male
birth_we	NCAPAPD	btwt_pn	Baby's birth weight	Defined by case
brth_wt	Alberta Vital Statistics/ PNOB	btwt_vs	Baby's birth weight	Defined by case

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
Birth and Infant Characteristics				
kind	Alberta Vital Statistics/ PNOB	kind_vs	kind of birth (Used to identify and eliminate multiple births and stillbirths)	10=Single live 11=Single stillbirth 20=Twins,both LB 21=Twins, 1LB,1SB 22=Twins, both SB 30=Triplets,all LB 31= Triplets, 2LB, 1SB 32=Triplets, 1LB, 2SB 33=Triplets, all SB 40=Quadruplets, all LB 41= Quadruplets, 3 LB, 1SB 42= Quadruplets, 2LB, 2SB 43= Quadruplets, 1LB, 3SB 44= Quadruplets, all SB 50= Quintuplets, all LB 51= Quintuplets, 4LB, 1SB 52= Quintuplets, 3LB, 2SB 53= Quintuplets, 2LB, 3SB 54= Quintuplets,1LB, 4SB 55= Quintuplets, all SB 99=unknown
multiple	NCAPAPD	kind_pn	Kind of birth: Multiple/Single (Used to identify multiple births)	0=single; 1=1st infant multiple; 2= 2nd infant multiple
ab	NCAPAPD	c_kind	Current multiple pregnancy (Used to identify multiple births)	0=absence 3=presence of risk
d1	NCAPAPD	def_pn	Fetal anomaly: current pregnancy (Used to eliminate anomalies)	0=absence 3=presence of risk

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
Maternal Age				
a	NCAPAPD	ag17	Age less than or equal to (LTE) 17 years	0=absence 1=presence
b	NCAPAPD	ag35	Age greater than or equal to (GTE) 35 years	0=absence 2=presence
Genetic or Constitutional				
c	NCAPAPD	wt91	Pre-pregnancy weight GTE 91 Kg	0=absence 1=presence
an	NCAPAPD	wtlt45	Pre-pregnancy weight LTE 45 Kg	0=absence 1=presence
d	NCAPAPD	htlt152	Pre-pregnancy height less than 152 cm	0=absence 1=presence
Obstetrical History				
term	NCAPAPD	term	Previous term births: Excludes Current	Defined by case
preterm	NCAPAPD	preterm	PTBs: Excludes Current	Defined by case
r	NCAPAPD	d2037	Delivery at 20 -37 weeks	0=absence 3= presence
aborta	NCAPAPD	abrt_pn	Abortions: Pregnancy prior to 20wks or 500g. Includes spontaneous and therapeutic abortions	Defined by case
abortion(96) aborts(97) aborts(98)	Alberta Vital Statistics/ PNOB	abrt_ob	Indicates number of abortions: includes ectopics	0-9 blank=missing
q	NCAPAPD	abrt1_pn	Previous abortions: 12-20 wks & under 500gms	0=absence 1=presence
p	NCAPAPD	nsb_pn	Stillbirths: after 20 weeks or wt 500 grams, no breathing	0= absence 3= presence
no_sbrth	Alberta Vital Statistics/ PNOB	nsb_vs	Number of Stillbirths	Defined by case
no_lbrth(96) no_lbrth(97) live(98)	Alberta Vital Statistics/ PNOB	live_vs	Live Births:Includes current	Defined by case
living	Alberta Vital Statistics/ PNOB	lvg_vs	Previous live births: Excludes current	Defined by case
O	NCAPAPD	ninfd_pn	Previous Neonatal Death	0= absence 3= presence

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
		Obstetrical History		
neoinfde	Alberta Vital Statistics/PNOB	ninfd_ob	Indicates number of neonatal or infant deaths (that mother has experienced)	0-9 blank=missing
s	NCAPAPD	prevcs	Previous cesarian section (C-S)	0=absence 2=presence
t	NCAPAPD	p_sga	Previous Small for Gestational Age	0=absence 2=presence
u	NCAPAPD	p_lga	Previous Large for Gestational Age	0=absence 3= presence
v	NCAPAPD	p_isou	Previous isoimmunization: unaffected infant	0=absence 1=presence
w	NCAPAPD	p_isoua	Previous isoimmunization: affected infant	0=absence 3=presence
x	NCAPAPD	p_def	Previous major anomaly	0=absence 2=presence
		Prenatal Care		
nprenatv	Alberta Vital Statistics/PNOB	nprenv	Number of times mother visited physician before delivery	00-30 # on form
attprenc	Alberta Vital Statistics/PNOB	prenc	Attendance at prenatal classes	1= no 2=yes blank=no response
		Lifestyle Factors		
e	NCAPAPD	smk_pn	Smoking during pregnancy	0=absence 2=presence
smoke	Alberta Vital Statistics/PNOB	smk_ob	Indicates whether mother smoked during pregnancy	1= no 2=yes 3=quit blank=no response
smokquan	Alberta Vital Statistics/PNOB	smkq	Smoking quantity	1=< 10 cigs/day 2=≥ 10cigs/day blank = no response
d3	NCAPAPD	alco_3	Alcohol ≥ 3 drinks (one occasion)	0=absence 3=presence
d4	NCAPAPD	alco_1	Alcohol ≥ 1 drink (per day)	0=absence 3=presence

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
		Lifestyle Factors		
usestrdr	Alberta Vital Statistics/PNOB	usestd	Indicates if mothers used street drugs	1= no 2=yes blank= no response
alcohol	Alberta Vital Statistics/PNOB	alco	Consumption of alcohol during pregnancy	1=no 2=yes blank = no response
alc_quan	Alberta Vital Statistics/PNOB	alcoq	Alcohol quantity during pregnancy	Text blank = no response
d5	NCAPAPD	druguse	Inappropriate or excessive drug use	0=absence 3=presence
drugfreq	Alberta Vital Statistics/PNOB	frestd	Frequency of drug use	Text blank= no response
		Pre-existing Medical Diseases		
f	NCAPAPD	diabd	Diabetes: Controlled diet	0=absence 1=presence
g	NCAPAPD	iddm	Insulin dependant diabetes	0=absence 3=presence
h	NCAPAPD	diabret	Diabetic retinopathy	0=absence 3=presence
i	NCAPAPD	hrta	Asymptomatic heart disease	0=absence 1=presence
j	NCAPAPD	hrts	Symptomatic heart disease	0=absence 1=presence
k	NCAPAPD	hypert	Chronic hypertension disease	0=absence 2=presence
l	NCAPAPD	hypertd	Anti-hypertensive drugs	0=absence 3=presence
m	NCAPAPD	renal	Renal disease	0=absence 2=presence
n	NCAPAPD	omedis	Other medical diseases (e.g., severe asthma, epilepsy, lupus, Crohn's disease)	0=absence 1=presence
		Nutritional Problems During Pregnancy		
ak	NCAPAPD	c_anem	Anemia (Hgb <100g/L)	0=absence 1=presence
am	NCAPAPD	por_wt	Poor weight gain (< 0.5kg/week or weight loss between 26-36 weeks)	0=absence 1=presence

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Original Variable	Data Source	New Name	Label Description	Values
Medical Problems During Current Pregnancy				
d2	NCAPAPD	acmedis	Acute medical problems (e.g., urinary tract infection, acute asthma, thyrotoxicosis)	0=absence 3=presence
aj	NCAPAPD	antib	Presence blood antibodies (e.g., RH, Anti-C, Anti-K, etc)	0=absence 3=presence
aa	NCAPAPD	c_poly	Polyhydramnios/ Oligohydramnios	0=absence 2=presence
ai	NCAPAPD	c_gestd	Gestational diabetes	0=absence 1=presence
al	NCAPAPD	preg41	Pregnancy ≥ 41 wks	0=absence 1=presence
ac	NCAPAPD	c_malp	Malpresentation	0=absence 3=presence
Pregnancy Complications				
ag	NCAPAPD	c_pih	Gestational Hypertension hypertension (PIH)	0=absence 2=presence
ah	NCAPAPD	c_prot	Proteinuria ≥ 1	0=absence 1=presence
ae	NCAPAPD	blt20	Bleeding < 20 weeks gestation	0=absence 1=presence
af	NCAPAPD	bgt20	Bleeding ≥ 20 weeks gestation	0=absence 3=presence
ad	NCAPAPD	c_memb	Membranes rupture prior to 37 weeks	0=absence 2=presence

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Original Variable	Data Source	New Name	Label Description	Values
Risk Scores				
ascore	NCAPAPD	ascore	Antepartum Risk Score	Case Score
iscore	NCAPAPD	iscore	Intrapartum Risk Score	Case Score
Intrapartum Risk Scoring				
I10	NCAPAPD	gest34	≥ 34 wks gestation	0=absence 2=presence
I11	NCAPAPD	gest356	35-36 wks gestation	0=absence 1=presence
I2	NCAPAPD	mecon	Meconium in labor	0=absence 1=presence
I3	NCAPAPD	iph_anem	Anemia	0=absence 1=presence
I4	NCAPAPD	fever	Fever	0=absence 1=presence
I5	NCAPAPD	fhrab	Fetal heart rate (FHR) abnormality	0=absence 1=presence
I6	NCAPAPD	ip_bld	Bleeding intrapartum	0=absence 1=presence
I7	NCAPAPD	rupmem24	Membranes ruptured > 24 hr	0=absence 1=presence
I8	NCAPAPD	seizure	Seizures	0=absence 1=presence
I9	NCAPAPD	coag	Coagulopathy	0=absence 1=presence
otheri	NCAPAPD	otheri	Comments	text

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
Indications for Induction				
signific	NCAPAPD	aph_ind	Antepartum hemorrhage	Y=yes N=no
intraute	NCAPAPD	infd_ind	Intrauterine death	Y=yes N=no
prom	NCAPAPD	rupt_ind	Premature rupture of membranes	Y=yes N=no
suspect	NCAPAPD	sga_ind	SGA	Y=yes N=no
ind_preg	NCAPAPD	pih_ind	Gestational hypertension:	Y=yes N=no
diabetes	NCAPAPD	diab__ind	Diabetes	Y=yes N=no
ind_gest	NCAPAPD	gestd	Gestational diabetes	Y=yes N=no
chronic_	NCAPAPD	chyp_ind	Chronic hypertension	Y=yes N=no
ind_othe	NCAPAPD	othe_ind	Other indications	text
Indications for Operative Delivery (op)				
fetal_di	NCAPAPD	fdist_op	Fetal distress	Y=yes N=no
intrapar	NCAPAPD	iph_op	Intrapartum hemorrhage	Y=yes N=no
placenta	NCAPAPD	prvia_op	Placenta previa	Y=yes N=no
maternal	NCAPAPD	hyper_op	Maternal hypertension	Y=yes N=no
maternal1	NCAPAPD	hrt_op	Cardiac disease	Y=yes N=no
maternal2	NCAPAPD	endo_op	Endocrine	Y=yes N=no
rh_isoim	NCAPAPD	iso_op	Rh isoimmunization	Y=yes N=no
fetal_ma	NCAPAPD	def_op	Fetal malformation	Y=yes N=no
fetal_il	NCAPAPD	fill_op	Fetal illness	Y=yes N=no
c-s-multi	NCAPAPD	cs_mult	C-S multiple pregnancy	Y=yes N=no
advanced	NCAPAPD	age_op	Advanced age	Y=yes N=no
c_s other	NCAPAPD	cs_oth	Comments	text

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
Additional Infant Information				
apgar_11	Alberta Vital Statistics/ PNOB	apg1_ob	Apgar 1	00-10 blank no response
apgar_5	Alberta Vital Statistics/ PNOB	apg5_ob	Apgar 5	00-10 blank no response
apgar_10	Alberta Vital Statistics/ PNOB	apg10_ob	Apgar 10	00-10 blank no response
apgar_1	NCAPAPD	apg1_pn	Apgar 1	Assigned
apgar_5	NCAPAPD	apg5_pn	Apgar 5	Assigned
apgar_10	NCAPAPD	apg10_pn	Apgar 10	Assigned
headcirc	Alberta Vital Statistics/ PNOB	headc	Head circumference	00=99 blank=missing
length	Alberta Vital Statistics/ PNOB	lgth	Length: crown to heel	00=99 blank=missing
fetal_an	NCAPAPD	fetal_an	Fetal anomaly: Resuscitation	Y=yes N=no
stillbir	NCAPAPD	rstilb	Stillbirth: Resuscitation No breathing, no heart beat	A= antepartum I=intrapartum
term	NCAPAPD	Term	Number of term births excludes current	Defined by case
gravida	NCAPAPD	gravida	Number of pregnancies	Defined by case
b_order	Alberta Vital Statistics/ PNOB	bord_vs	Birth order for current pregnancy	1= single number for multiple
b_place	Alberta Vital Statistics/ PNOB	bplac_vs	Birth place (96) Birth place (97) Birth place (98)	1=hospital 2=enroute 3=nursing home 4=other 5=at home 9=unknown
b_place location	Alberta Vital Statistics/ PNOB	bplac_vs	Birth place (97) Birth place (98)	1=hospital 2=at home 3=enroute 4=auxiliary hosp 5=nursing home 6=unknown 7=other 9=Hospice

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
Additional Birth Information				
b_attend	Alberta Vital Statistics/ PNOB	attb_vs	Birth attendant	1996 1=physician 2=registered nurse 3=midwife 5=other 9=unknown
b_attend	Alberta Vital Statistics/ PNOB	attb_vs	Birth attendant	1997/8 1=physician 2=registered nurse 3=midwife 5=other 6=unknown
Marital Status				
m_marrrst	Alberta Vital Statistics/ PNOB	marrst_vs	Marital Status	1996 1=single 2=married 3=widowed 4=divorced 5=separated 9=unknown
m_marrrst	Alberta Vital Statistics/ PNOB	marrst_vs	Marital Status	1997/8 1=never married 2=married 3=widowed 4=divorced 9=unknown

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
		Geographic Information		
sgc	Alberta Vital Statistics/ PNOB	sgc_vs	Geographic code birth	Defined by case
pl_rha	Alberta Vital Statistics/ PNOB	brha_vs	Health Region where birth occurred	01=Chinook 02=Palliser 03=Headwaters 04=Calgary 05= HA#5 06=David Thompson 07=East Central 08= Westview 09=Crossroads 10=Capital 11=Aspen 12=Lakeland 13=Mistahia 14=Peace 15=Keeweetinok 16=Northern Lights 17=Northwestern 90=unknown
postcode	Alberta Vital Statistics/ PNOB	pc_vs	Postal code	Defined by case
m_resid	Alberta Vital Statistics/PNOB	mres_vs	SGC of usual residence	Defined by case
r_rha	Alberta Vital Statistics/PNOB	rrha_vs	Health region of mothers residence	01=Chinook 02=Palliser 03=Headwaters 04=Calgary 05= HA#5 06=David Thompson 07=East Central 08= Westview 09=Crossroads 10=Capital 11=Aspen 12=Lakeland 13=Mistahia 14=Peace 15=Keeweetinok 16=Northern Lights 17=Northwestern 90=unknown

Assembled File: NCAPAPD and the Combined Alberta Vital Statistic and PNOB

Variable Original	Data Source	New Name	Label Description	Values
		Geographic Information		
phsarean	Alberta Vital Statistics/ PNOB	phsareas	Public Health Service Areas (PHSA)	01= St Alberta 02=Castledowns 03=West Central 04=Central 05= North Central 06=North East 07=West 08= West (South) 09=South West (West) 10=South West (East) 11=South Central 12=Millwoods 13=Sherwood Park 14=Strathcona County 15=Thorsby 16=Leduc 17=Beaumont blank= other
		Socio-Economic Attributes		
cfinc	Census	cfinc	Median Census Family Income (median census incomes were derived for 17 PHSAs within CHA). Each woman was initially assigned the income value for the PHSA.	Defined by case
lessgr9	Census	lessgr9	Less Than Grade 9 Education Groupings (% of less than grade 9 education were derived for 17 PHSA within CHA)	Defined by case
bacdeg	Census	bacdeg	Baccalaureate Degree or Greater (% baccalaureate degree or greater were derived for 17 PHSA within CHA).	Defined by case
sparent	Census	sparent	Single Parent Grouping (% single parent were derived For 17 PHSA within CHA).	Defined by case
aborigin	Census	aborigin	Aboriginal Population Grouping (%Aboriginal Population Were Derived For 17 PHSA within CHA).	Defined by case

Appendix C: Data Transformations and Data Combinations

Data Transformations and Data Combinations

Birth Weight, Gestational Age, and Birth Weight

Maternal age (mage_pn, mage_vs), infant birth weight (btwt_pn, btwt_vs), and gestational age (gest_pn, gest_vs) were available from both the NCAPAPD and the PNOB database. The PNOB versions were utilized for each of the maternal age and birth weight variables because one or more cases were missing or were identified as a 0 in the NCAPAPD. Each of these variables was then used to develop cut points for low birth weight (btwsvslbw), maternal age risk categories (magevsgp) and preterm or term birth (gestvspt).

Low birth weight (btwvslbw) for the purpose of this study was categorized as less than 2500 grams. Although this categorization is consistent with the research literature it does not address the 'heterogeneity' of low birth weight. That is, an infant may be low birth weight (< 2,500 grams) due to: 1.) a normal growth rate but a shortened gestational period (Preterm/ Appropriate for Gestational Age (P-AGA)); 2.) an inadequate growth rate and a shortened gestational period (Preterm/Small for Gestational Age (P-SGA)); and 3.) an inadequate growth rate during a normal gestational age (Term/Small for Gestational Age (T-SGA). The low prevalence of several risk factors and LBW infants (n=1,269) did not permit such analysis. Consequently, it was not possible within this study to specifically differentiate between preterm delivery (P-AGA) and growth restriction (P-SGA and T-SGA). To examine the impact of the numerous risk factors on PTB, gestational age (gestvspt) was differentiated into PTB (<37 weeks) and term birth (≥37 weeks).

The maternal age categories (magevsgp) were 12-19 years of age (representing young mothers), 20-34 years (representing the reference group), and 35-47 years of age (representing older mothers). The younger mother and older mother risk categories were developed based on comparative categorization in existing research literature.

Type of Birth: Multiple Births, Singleton Births, and Stillbirths

Type of birth (kind_vs, kind_pn, c_kind) was included in the NCAPAPD database and the PNOB database. The variable kind_vs provided more specific data on the type of birth, permitting closer examination of the data with respect to the type of single birth (stillbirth or live) and the type of multiple birth (stillbirth or live). In contrast, c_kind indicated if the birth was multiple or single, and kind_pn indicated if the birth was single or if the infant of the multiple birth was the first or second infant of the multiple birth. Type of birth (kind_vs) was used in the examination of and elimination of multiple births and stillbirths.

Obstetrical History

Stillbirth (nsb_vs, nsb_pn), previous livebirths (lvg_vs, lvg_pn), previous abortions (abrt1_pn, abrt_pn, abrt_ob), and previous neonatal deaths (ninfd4_pn, ninfd_ob) were four obstetrical history variables found in the NCAPAPD and PNOB database. The variable abrt_pn was selected because it provided a more comprehensive definition of previous abortions, including both spontaneous and therapeutic abortions. Both abrt1_pn and abrt_ob did not have this differentiation and abrt_ob had one or more cases in which the data were missing. The abrt_pn variable was recoded (nabrt) to address the categories 0 (no previous abortions) and 1 (1or more abortions). This differentiation was to allow for comparisons with several other studies that indicated that there is an increased risk for LBW and PTB with one or more abortions (Lumley, 1993).

Previous stillbirth (nsb_vs) from the PNOB database was selected because it was possible to quantify the numbers of stillbirths. It was recoded (nsbfin) into two categories (0=no stillbirths; 1=one or more stillbirths). Previous neonatal death (ninfd4_pn) from the NCAPAPD database was selected for analysis because the data were more complete. Lastly, previous live births (lvg_vs_) was selected from the PNOB database and recoded as (prtyrec) with 0 births =1, 1-3 births equal to 0, and 4 or greater births equal to 2.

Nulliparous women have been identified as being at an increased risk for poor birth outcomes. Consequently, they were identified as the at risk category group. Three parity groups were developed to provide descriptive information about parity. In addition previous live births (lvg_vs_) was coded (prtyrec2) describing women according to nulliparous (coded 1) and multiparous status (coded 0). Nulliparous indicated that women did not have a previous livebirth and multiparous indicated that women had one or more previous livebirths. In the MLR models nulliparity and multiparity were used as multiparity is considered to be a more favorable outcome (Kramer, 1987) and nulliparity has been found to have a slightly increased risk of an adverse birth outcome (Lumley, 1993; Berkowitz, Blackmore-Prince, Lapiniski, & Savitz, 1998).

Pre-Existing Diseases and Complications of Pregnancy

Data combinations were also possible within one database. Within the NCAPAPD database, there were 3 the pre-existing diabetes variables: diabetes diet controlled (diabd), insulin depedent diabetes mellitus (iddm) and diabetic retinopathy (diabret). There were also 2 variables that referred to heart disease: asymptomatic heart disease (hrta) and symptomatic (hrts), and 2 chronic hypertension variables: chronic hypertension (hypert) and chronic hypertension and drug controlled (hypertd). Due to the small numbers of women with pre-existing diseases the different categorizations of each disease were combined. That is, for each of these pre-existing diseases the different categorizations for each disease were combined to create one singular variable for each disease. For example, using the rule that if diabd or iddm or diabret =1, then (diabdis)=1 (presence of diabetes) and if diabd or iddm or diabret=0 then diabdis=0 (absence of disease). This rule was followed for both heart disease (hrtdis) and hypertensive disease (hypertrec).

To create a new variable (gestational hypertension with proteinuria: pre-eclampsia), the gestational hypertension variable (c_pih) and the proteinuria greater than or equal to one variable (c_prot) were combined, creating this new variable (eclamp). The same combination rule that was used for the creation of the pre-existing medical diseases was also used to create the new gestational hypertension with proteinuria variable. Creation of this new variable permitted the creation of a variable that indicated the severity of gestational hypertension.

Smoking Status and Alcohol Status

An advantage of using linked databases is the ability to combine variables either to augment or enrich the data or to broaden the conceptualization of a variable. In this study the alcohol data and the smoking data from both databases were combined. Thus, it was possible to identify mothers who had quit smoking during their pregnancy as well as the smokers and nonsmokers. The variable smk_ob from the PNOB database provides information about whether the mother was a smoker, non- smoker, or quit smoking during her pregnancy, while the variable smk_pn variable provides data about whether the mother was a smoker or nonsmoker. To differentiate between smokers, nonsmokers and quitters variables, were transformed to create new variables. Two variables were combined into two new variables; one variable that represented smokers (nsmoke2) and one variable that represented quitters (quitters2). The new variable smokers (nsmoke2) indicated that if either the PNOB database or the NCAPAPD indicated that the woman was a smoker, the variable was assigned a one otherwise a value of 0 was assigned (representing non-smokers). For the variable quitters (quitters 2), if smk_ob=2 then quitters 2=1 represented quitters.

There were two alcohol status variables (alco_1, alco_3) in the NCAPAPD database and one alcohol variable (alco) in the PNOB database. Both alco_1 and alco_3 measured frequency of drinking, one drink per day and three or more drinks on any one occasion, whereas alcohol consumption (alco) in the PNOB database was measured as presence or absence. The three variables were combined into one variable (nalco) according to the rule that if alco_1 or alco_3 or alco =1 then nalco=1 (drank) and if alco_1or alco_3 or alco=0 then nalco=0 (did not drink).

Missing Data Transformations: Use of Street Drugs, Prenatal Classes and Prenatal Visits

The last data transformations to be discussed include use of street drugs, prenatal classes, and number of prenatal visits. These three variables were captured solely in the PNOB database. However, the street drug use, prenatal classes and prenatal visit variables had a substantial amount of missing data. In the final data set (n=26,265), 3.8%

(n=1,005) of the women had missing street drug use data, 10.4 % (n=2,730) had missing prenatal visits data, and 14.1% (n=3,686) had missing prenatal class data.

In general, analytic programs delete all cases with missing data prior to analysis. Many schemes have been proposed to avoid losing all of the data from subjects missing data on one or more of a small number of variables. The technique used here was proposed by Cohen & Cohen (1984) and involves creating a new variable for each variable for which data are missing. This variable is assigned a value of 1 if the data are missing for this subject; otherwise, the data are assigned a 0. A value is also assigned to the original variable for all data that were missing. Often, in analysis involving continuous variables, this value is the mean. With a binary variable, either a zero or a one is chosen, often to enhance interpretability of the final analysis.

In general, it was hypothesized that individuals with missing data would be more similar to individuals with the less desirable value (behavior). Consequently, individuals with missing data and individuals with the risk behavior were assigned a one in the original variable and women without the risk behavior were assigned a zero. In the new variable (data missing variable) the subjects with missing data were assigned a one, otherwise a zero was assigned. If the hypothesis is correct, the new variable for the missing data in the logistic regression should not be significant, indicating that the women with missing data were in fact more like the women who engaged in a risk behavior. As a result of development of a missing variable for those women who had missing data for the specific index variable, there were two variables representing each construct, a missing variable and the index variable.

For example, in the case of the prenatal classes variable (prenc) there were two variables, one representing women with missing data (classmiss) and a variable identifying attendance or non-attendance (mclass). It is important to note that the initial coding of prenc was not conventional in that the value 0 identified women who did not attend prenatal classes and the value 1 indicated that women attended prenatal classes. As a result of this initial coding, the coding convention for the missing variable (classmis) and the new attendance and non-attendance variable needed to change. For the new prenatal class index variable (mclass) the women with missing classes and the women who did not attend prenatal classes were assigned a 0, and the women who attended prenatal classes were assigned a 1. For the missing variable (classmiss) women who attended classes and did not attend classes were assigned a numerical value of 0, and the women who had missing data were assigned a 1, representing the risk category. It is also recognized that an alternative hypothesis may be that attention to recording of prenatal classes and number of prenatal visits may be minimal following birth; thus the missing data is a non-response by the health provider.

There is extensive debate in the literature about the adequate number of visits for optimal follow-up and care and the most appropriate measure to capture adequacy of prenatal visits. A number of investigators believe that measurement must include a gestational age adjustment and should account for when the prenatal care was initiated (Alexander & Kotelchuk, 1996; Koroukin & Rimm, 2001; Kotelchuk, 1994). Initiation of prenatal care (i.e., month prenatal care was initiated) was not recorded in the PNOB database. Thus the Kessner and Kotelchuk index could not be utilized.

The same technique of creating a new variable for the missing prenatal visit was utilized for women with missing data for prenatal visits. There were three new variables that represented prenatal visits. There was a missing variable (prenamis), a variable representing fewer than or equal to 4 visits (mprena), and a variable representing 5 to 7 visits (vis57). If a woman had four or fewer visits (nprenv $\leq$4), then mprena was equal to 1. Similarly, if a woman had missing data (nprenv = missing) then mprena was equal to 1. For women who had greater than 4 visits (nprenv = >4) mprena was equal to 0. For the missing variable (prenamis) women with missing visits were assigned a 1; otherwise women were assigned a 0. Lastly, for the prenatal visits variable vis 57, women with 5-7 visits (nprenv = 5$\leq$ 7) were assigned a 1; otherwise women were assigned a 0. The categorizations of visits were chosen based on existing research which has included multicentre randomized controlled trials whereby new models of reduced number of visits (visits schedules ranging from 4-9) were compared to standard model of antenatal visits. (Carroli, Villar, Piaggo, Khan-Neelfour, Gulmeaoglu, 2001; Villar, Ba'aqueel, Piaggio, Lumbiganon, Belzian et al, 2001). In the Carroli et al. (2001) WHO systematic review of seven randomized antenatal care control trials of 57,418 women, there were no clinically differential effects of the reduced number of antenatal visits when the results were pooled for low birth weight (pooled estimateOR=1.04, CI: 0.93-1.17) and other maternal outcomes.

The Cohen and Cohen procedure (1984) of creating a new variable for each for missing data is known to be biased (Schopflocher, D., personal communication, April 2002). However, alternative procedures such as multiple imputation (Schopflocher, D., personal communication, April, 2002) will also be biased and perhaps very severely biased in the event that the probability that data will be missing is dependent on the actual value of the variable (i.e., if people with very few visits are also more likely to have missing values).

Limitations are apparent in all indices of adequacy of prenatal care as they do not adjust for the complicated pregnancy that requires more visits. Consequently, there will be women who may have been identified as having adequate care (increased number of visits) but the outcome was adverse (preterm). Thus, the measure will indicate that with increased visits a poor outcome still existed. A potential partial solution that might capture some of these problems would be to have interaction variables between the complications and the index variables.

A second limitation of the indices is the apparent concentration of visits (e.g., one third approximately in the last 8 weeks of the pregnancy). This concentration of visits may bias the guidelines themselves, in that if women deliver prior to term then the reduced number of visits is associated with the poor outcome. In addition, there needs to be a methodological way of weighting the visits for high risk pregnancies that require more visits because this increased utilization also can also be associated with a poor outcome.

Geographic Data Combinations

A key component of this study was to examine the influence of socio-economic variables on the outcome of interest (i.e., LBW and PTB). Consequently, it was important both to confirm that mothers who gave birth in a specific RHA were also residents of that RHA, and to reassign women with a missing birth regional health authority to their residential RHA. To complete this transformation the residential RHA variable (rrha_vs), birth RHA variable (brha_vs), PHSA variable (phsareas), and the RHA boundary variable (RHA98) that identifies the geographic boundaries of each RHA were used. This data transformation made it possible to select only those births in CHA (RHA10) and to reassign births to the remaining RHAs. Specific to CHA, it was also possible to identify the seventeen PHSA and those women (n=1,387) who were residents and gave birth in CHA but for whom there was no allocated PHSA.

To assign births in the RHAs that were resident in other RHAs (i.e., RHAs 1-5 or 6-17) and to identify RHAs according to the RHA1998 boundary, the variable *brha2* was created. Thus, if a woman had given birth in CHA (RHA 10) but her residential RHA was Calgary Health Region (RHA 4) then the birth was designated to the residential birth region of Calgary. The variable brha2 was then recoded to identify RHAs 1-5. Following this step the variable *brha3* was created to identify the births within the PHSAs of CHA (RHA 10) and to uniquely identify the remaining RHAs. This transformation was created by means of the following rules: 1.) if brha2 =10 assign the PHSA (phsarea); 2.) if the PHSA (phsarea was missing (phsarea=0) then brha3=18; 3.) if brha2 is unknown (brha2=90) then recode to brha3=97; and 4.) if brha2 =1,6, 7, 8,9,11,12,13,14,15,16,or 17 then assign 100 (i.e.,brha2 +100) to each birth RHA. To facilitate selection of only the PHSA (1-18) the brha3 variable was recoded into a new variable allareas2.

Census Data Transformations

As noted previously, each of the 17 PHSAs had its own derived median census family income (cfinc), percentage of aboriginal population (aborigin), percentage of single parent families (sparent), percentage of persons with less than grade nine education (lessgr9), and percentage of persons with baccalaureate education or greater (bacdeg). As a result, each woman within each PHSA was assigned the value of her respective PHSA. Based on the range of each of these variables cutpoints were developed for each variable. Median census family income (cfinc) was recoded into a new variable (cfincgrp) with four categories: cfincgrp 60-69999=0, cfincgrp 50-59999 =1, cfincgrp 40-49999=2, cfinc 30-39999=3. For the variables education (lessgr9, bacdeg) percentage of single parent families (sparent), and percentage of aboriginal population (aborigin), low, moderate and high risk categories were created based on a natural breaks in the data points. The numerical value 0 indicates low risk, 1 indicates moderate risk, and 2 indicates a high risk. The risk groupings were acknowledged in the recoding of these variables (gr9grp, bacdeggrp, spargp, aborgrp).

Further transformations were completed for the census data. The above transformations still represent a large number of variables in relation to the total sample size of 17 PHSAs. Consequently, it was decided that it was necessary to explore the relationships between these variables to consider the possibility that they represent a smaller number of concepts.

Examination of the correlation matrix in Table 16 shows many extreme values and suggests that the effectiveness of a regression analysis would be greatly reduced due to collinearity.

Table 16
Correlation Matrix of Socio-economic Variables

Variable	cfinc	sparent	aborigin	lessgr9	bacdeg
cfinc	1.000	-.4196	-.8099**	-.8724**	.5578*
sparent	-.4196	1.000	.5641*	.4257	.3021
aborigin	-.8099**	.5641*	1.000	.8059*	-.4110
lessgr9	-.8724**	.4257	.8059**	1.000	-.5612*
bacdeg	.5578*	.3021	-.4110	-.5612*	1.000

* denotes Significant ≤ .05 * *denotes Significant ≤ .01 (2 –tailed)

As a result of the findings in the correlation matrix, a principal component analysis was undertaken to determine whether these 5 variables could be replaced by a smaller number of linear combinations of these variables. That is, could the observed variables be explained in terms of a smaller number of variables called factors. The principal component analysis indicated that two factors were sufficient to capture the interrelationships among these 5 variables to a large degree. The first two factors accounted for 91.1% of the variability amongst the 5 variables. The factor loadings are shown in Table 17.

Table 17
Principal Component Analysis

Variable	Factor 1	Factor 2
cfinc	-.94505	.06043
sparent	.51360	.82752
aborigin	.92103	.14931
lessgr9	.94548	-.05900
bacdeg	-.57464	.78248

The loadings of variables on the factors represent correlations of each of the variables with a hypothetical underlying factor. Examining the pattern of these loadings can allow the analyst to make inferences about the nature of the underlying factor. Thus, Factor 1 is interpreted as "Low Socio-Economic Status" because the median census family income correlates strongly and negatively and so does the proportion of people who have higher education. Conversely, the proportion of people who are Aboriginal, have a low educational attainment and are single parent families correlate positively, as we would have expected under this interpretation.

The second factor is more difficult to understand. It is associated with both higher educational status and higher proportions of single parent families. It was thought to represent the characteristics of resourcefulness and stability. The census definition of single parent families (lone parent) may assist in the interpretation. Single parent families refers to a father or mother, with no common law or spouse present, living in a dwelling with one or more never-married sons and/or daughters (blood, step, or adopted). It might be that this finding is a unique characteristic representing resourceful career-focused single parents, or this finding may also be a unique characteristic of stability and cohesion in the community.

The principal component procedure allows a score on each factor to be derived for each of the PHSA (as an additive combination of the scores from all variables in the analysis). These scores were calculated and assigned to each individual.

The following tables provide the specific information on the data transformations and the combining of variables.

Data Transformations and Data Combinations

Variable Original (Data Assemble Variable Name)	Data Source	Variable Name	Label Description	Values
Birth and Infant Characteristics				
sex (sex_vs)	Alberta Vital Statistics /PNOB	sex_vs	Baby's Sex	1=male 2=female
sex (sex_pn)	NCAPAPD	sex_pn	Baby's Sex	1=male 2=female 9=missing
Gestatio (gest_vs)	Alberta Vital Statistics /PNOB	gestvspt	Preterm/Term Birth	0= term ≥ 37weeks 1=preterm < 37 weeks
brth_wt (btwt_vs)	Alberta Vital Statistics /PNOB	btwvslbw	Low birth weight	0=≥ 2500 gms 1=< 2500 gms
d1 (def_pn)	NCAPAPD	def_pn	Fetal anomaly:current pregnancy (Used to examine anomaly births)	0=absence 1=presence
ab (c_kind)	NCAPAPD	c_kind	Current multiple pregnancy (Used to examine multiple births as a secondary source)	0=absence 1=presence
Stillbir (rstilb)	NCAPAPD	rstilb	Stillbirth: No breathing, no heart beat (Used to examine stillbirths as secondary source)	1= antepartum 2=intrapartum
Maternal Age				
mother_a (mage_vs)	Alberta Vital Statistics /PNOB	magevsgp	Maternal Age Risk Group	0=20-34 years 1=12-19 years 2=≥35years

Data Transformations and Data Combinations

Variable Original (Data Assemble Variable Name)	Data Source	Variable Name	Label Description	Values
Obstetrical History				
preterm (preterm)	NCAPAPD	npreterm	Previous PTBs	0= absence 1= 1 or more
living (lvg_vs)	Alberta Vital Statistics/ PNOB	prtyrec	Parity: number of previous live births	0= 1-3 births 1= 0 births 2=4 or greater births
living (lvg_vs)	Alberta Vital Statistics/ PNOB	prtyrec2	Parity: number of previous live births	0=multiparous (1 or more) 1=primiparous (no previous birth)
aborta (abrt_pn)	NCAPAPD	nabrt	Abortions: Pregnancy prior to 20 wks or 500 gms Includes SA & TA	0=absence 1= 1 or more
no_sbrth (nsb_vs)	Alberta Vital Statistics/ PNOB	nsbfin	Number of stillbirths	0= absence 1= 1or more
o (ninfd_pn)	NCAPAPD	ninfd4pn	Previous neonatal death renamed to do data check	0=absence 1=presence
t (p_sga)	NCAPAPD	p_sga	Previous SGA	0=absence 1=presence
Pre-existing Medical Diseases				
f (diabd)	NCAPAPD	diabd	Diabetes: Diet (Used to combine variable)	0=absence 1=presence
g (iddm)	NCAPAPD	iddm	Insulin dependant diabetes (Used to combine variable)	0=absence 1=presence
h (diabret)	NCAPAPD	diabret	Diabetic Retinopathy (Used to combine variable)	0=absence 1=presence
fgh (diabd) (iddm) (diabret)	NCAPAPD	diabdis	Diabetes (Combined variable name= diabd=1/iddm=1/or diabret=1 then diabdis=1; diabd=0/iddm=0/or diabret=0 then diabdis=0	0= absence 1=presence
i (hrta)	NCAPAPD	hrta	Asymptomatic heart disease (Used to combine variable)	0=absence 1=presence
j (hrts)	NCAPAPD	hrts	Symptomatic heart: disease (Used to combine variable)	0=absence 1=presence

Data Transformations and Data Combinations

Variable Original (Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Pre-existing Medical Disease				
i,j (hrta) (hrts)	NCAPAPD	hrtdis	Heart disease** (Combined variable= hrta=1/hrts =1 then hrtdis=1; hrta=0/hrts =0 then hrtdis=0)	0=absence 1=presence
k (hypert)	NCAPAPD	hypert	Hypertension (Used to combine variable)	0=absence 1=presence
l (hypertd)	NCAPAPD	hypertd	Anti-hypertensive drugs (Used to combine variable)	0=absence 1=presence of risk
k,l (hypert) (hypertd)	NCAPAPD	hyprtrec	Chronic hypertension (Combined variable name= hypert=1/hypertd =1then hyprtrec=1; hypert=0/hypertd =0 then hyprtrec=0)	0=absence 1= presence of risk
m (renal)	NCAPAPD	renal	Renal disease	0=absence 1=presence
Medical Problems During Current Pregnancy				
d2 (acmedis)	NACPOD	acmedis	Acute medical problems during pregnancy	0=absence 1=presence
aa (c_poly)	NCAPAPD	c_poly	Polyhydramnios/ Oligohydramnios	0=absence 1=presence
ai (c_gestd)	NCAPAPD	c_gestd4	Gestational diabetes during current pregnancy (c_gestd changed to c_gestd4 for recheck on data)	0=absence 1=presence

Data Transformations and Data Combinations

Variable Original (Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Pregnancy Complications				
ad (c_memb)	NCAPAPD	c_memb	Membranes rupture prior to 37 weeks in current pregnancy	0=absence 1=presence
Af (bgt204)	NCAPAPD	bgt204	Bleeding ≥ 20 weeks gestation in current pregnancy (bgt20 changes to bgt204 for recheck on data)	0=absence 1=presence
ag (c_pih)	NCAPAPD	c_pih	Gestational Hypertension in current pregnancy Used to combine variable	0=absence 1=presence
ah (c_prot)	NCAPAPD	c_prot	Proteinuria ≥ 1 in current pregnancy (Used to combine variable)	0=absence 1=presence
ag, ah (c_pih) (c_prot)	NCAPAPD	eclamp	Gestational Hypertension with Proteinuria (Combined c_pih=1& c _prot=1 then eclamp=1; c-pih=0 & c-prot=0 then eclamp=0;	0= absence 1=presence
placenta (prvia_op)	NCAPAPD	previa	Placenta previa in current pregnancy	0= absence or no identification of previa 1=presence

Data Transformations and Data Combinations

Variable Original Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Lifestyle Factors				
e (smk_pn)	NCAPAPD	smk_pn	Smoking during pregnancy (Used to combine variable)	0=absence (no) 1=presence (yes)
smoke (smk_ob)	Alberta Vital Statistics /PNOB	smk_ob	Indicates whether mother smoked during pregnancy (Used to define quitters)	0=no 1=yes 2=quit
e, smoke (smk_pn) (smk_ob)	NCAPAPD Alberta Vital Statistics /PNOB	nsmoke2	Smoking (Combined smk_pn=1, smk_ob=1 then nsmoke2=1; smk_pn=0, smk_ob=0 then nsmoke2=0	0=nonsmokers & quitters 1=smokers
smoke (Smk_ob)	Alberta Vital Statistics /PNOB	quitters2	Quitters (Combined smk_ob=2 then quitters2=1; quitters=0 smokers and non smokers)	0=otherwise 1=quitters
d3 (alco_3)	NCAPAPD	alco_3	Alcohol ≥ 3drinks (one occasion) (Used for combined variable nalco)	0=absence (no) 1=presence (yes)
d4 (alco_1)	NCAPAPD	alco_1	Alcohol ≥ 1 (per day) (Used for combined variable nalco)	0=absence (no) 1=presence (yes)
alcohol (alco)	Alberta Vital Statistics /PNOB	alco	Consumption of alcohol during pregnancy (Used for combined variable nalco)	0=absence (no) 1= presence (yes)
d3, d4, alcohol (alco_3), (alco_1) (alco)	NCAPAPD Alberta Vital Statistics /PNOB	nalco	Alcohol (Combined alco_3=1 /alco_1=1 /alco=1 then nalco=1; alco_3=0 /alco_1=0 /alco=0 then nalco=0	0= absence (no) 1= presence (yes)
usestrdr (usestd)	Alberta Vital Statistics /PNOB	usestd	Indicates if mothers used street drugs. Also women with missing data represented with a 1	0= no use 1=use
usestrdr (usestd)	Alberta Vital Statistics /PNOB	musestd	Missing variable street drug use for missing data Missing used street drugs initially coded as .33 then recoded to 1.	0=users and non users 1=missing
d5	NCAPAPD	druguse	Inappropriate or excessive drug use during pregnancy	0=absence 1=presence

Data Transformations and Data Combinations

Variable Original (Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Prenatal Care				
nprenatv (nprenv)	Alberta Vital Statistics /PNOB	nprenv	Number of times mother visited physician before delivery.	00-30 # on form
Nprenv (nprenv2)	Alberta Vital Statistics /PNOB	nprenv2	Number of times mother visited physician before delivery (creation 2nd variable for recheck and SOGC)	00-30 # on form
nprenv (mprena)	Alberta Vital Statistics /PNOB	mprena	Prenatal visits $\geq$ 4 Also if nprenv =missing then mprena was =1	0=GT 4 visits 1=LTE 4 visits
nprenv (mprena)	Alberta Vital Statistics /PNOB	prenamis	Missing variable prenatal visits. Missing initially coded as .18 then recoded to 1.	0= otherwise 1= missed visits
nprenatv (nprenv)	Alberta Vital Statistics /PNOB	vis57	Prenatal visits 5-7 was computed to extend the number of visits	0=other visits 1=5-7 visits
attprenc (prenc)	Alberta Vital Statistics /PNOB	prenc	Attendance at prenatal classes	0= no 1=yes blank=miss
prenc	Alberta Vital Statistics /PNOB	mclass	Prenatal classes women with missing classes also were a 1	0=no attend 1= attend
prenatal classes (prenc)	Alberta Vital Statistics /PNOB	classmis	Missing Prenatal Classes Missing initially coded as .22 then recoded to 1.	0=otherwise 1= missed classes

Data Transformations and Data Combinations

Variable Original (Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Geographic Information				
r_rha (rrha_vs)	Alberta Vital Statistics	rrha_vs	Health region of mothers residence	01=Chinook 02=Palliser 03=Headwaters 04=Calgary 05= HA#5 06=David Thompson 07=East Central 08= Westview 09=Crossroads 10=Capital 11=Aspen 12=Lakeland 13=Mistahia 14=Peace 15=Keeweetinok 16=Northern Lights 17=Northwestern 90=unknown
pl_rha	Alberta Vital Statistics	brha_vs	Health Region where birth occurred	01=Chinook 02=Palliser 03=Headwaters 04=Calgary 05= HA#5 06=David Thompson 07=East Central 08= Westview 09=Crossroads 10=Capital 11=Aspen 12=Lakeland 13=Mistahia 14=Peace 15=Keeweetinok 16=Northern Lights 17=Northwestern 90=unknown
RHA98	Alberta Vital Statistics	RHA 98	Boundary for RHA's in Alberta	01=Chinook 02=Palliser 03=Headwaters 04=Calgary 05= HA#5 06=David Thompson 07=East Central 08= Westview 09=Crossroads 10=Capital; 11=Aspen

Data Transformations and Data Combinations

Variable Original (Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Geographic Information				
RHA98	Alberta Vital Statistics	RHA 98	Boundary for RHA's in Alberta Continued	12 Lakeland 13=Mistahia 14=Peace 15=Keeweetinok 16=Northern Lights 17=Northwestern 90=unknown
phsarean	Alberta Vital Statistics	phsareas	Public Health Service Areas (PHS areas) of Capital Health	01= St Albert 02=Castle Downs 03=West Central 04=Central 05= North Central 06=North East 07=West 08= West (South) 09=South West (West) 10=South West (East) 11=South Central 12=Millwoods 13=Sherwood Park 14=Strathcona County 15=Thorsby 16=Leduc 17=Beaumont blank=0
pl_rha, RHA 98 (brha_vs, RHA98)	Alberta Vital Statistics	brha2	To assure that the RHA 98 boundary was applied to the identified births brha2 reassigned the births within each of the brha. The brha2 variable was recoded to assign RHA's (southern RHA to one value 01). Also those women who gave birth in one RHA but their residence was another RHA the birth was assigned to the residential RHA	01=South RHAs 06=David Thompson 07=East Central 08= Westview 09=Crossroads 10=Capital 11=Aspen 12=Lakeland 13=Mistahia 14=Peace 15=Keeweetinok 16=Northern Lights 17=Northwestern 90=unknown RHAS

Data Transformations and Data Combinations

Variable Original (Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Geographic Information				
pl_rha, r_rha phsarean RHA 98 (brha_vs, rrha_vs, RHA982 phsareas)	Alberta Vital Statistics	brha3	Births in birth RHA assigned to residential rha. Includes births in Capital Health without assigned Public Health Service Area (brha and r_rha=10). Using the brha2 variable those births in Capital Health that did not have a Public Health Service Area were assigned the number 18. To identify the the southern RHAs and RHA 6-9,11-17, the number 100 was added	01= St Albert 02=Castle Downs 03=West Central 04=Central 05= North Central 06=North East 07=West 08= West (South) 09=South West (West) 10=South West (East) 11=South Central 12=Millwoods 13=Sherwood Park 14=Strathcona County 15=Thorsby 16=Leduc 17=Beaumont 18 =Missing PHSA 101=South RHAs 106= RHA 6 107=RHA 7 108=RHA 8 109= RHA 9 111= RHA 11 112=RHA 12 113=RHA 13 114= RHA14 115= RHA 15 116= RHA 16 117= RHA17 97=Missing RHA
pl_rha, r_rha phsarean rha98 (brha_vs, rrha_vs, phsareas,)	Alberta Vital Statistics	allareas2	Public Health Service Areas within Capital Health identified separately from the variable brha3	01= St Albert 02=Castle Downs 03=West Central 04=Central 05= North Central 06=North East 07=West 08= West (South) 09=South West (West) 10=South West (East) 11=South Central

Data Transformations and Data Combinations

Variable Original (Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Geographic Information				
pl_rha, r_rha phsarean rha98 (brha_vs, rrha_vs, phsareas,)	Albert Vital Statistics	allareas2	Includes births of mothers who were residents of CHA Capital Health (Includes Births that occurred in Capital Health, reassigned births of mothers who were residents of Capital Health)	12=Millwoods 13=Sherwood Park 14=Strathcona County 15=Thorsby 16=Leduc 17=Beaumont 18 =RHA 10 Missing pc to identify to PHS Area
Interactions				
c_pih, magevsgp	NCAPAPD & Alberta Vital Statistics PNOB	c_pih* magevsgp	Gestational Hypertension by Maternal Age	
mclass, prtyrec2	NCAPAPD & Alberta Vital Statistics/ PNOB	mclass* prtyrec2	Prenatal Classes by Parity	
nalco, nsmoke2	NCAPAPD & Alberta Vital Statistics/ PNOB	nalco* nsmoke2**	Alcohol by Smoking	

Data Transformations and Data Combinations

Variable Original Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Socio-Economic				
cfinc	Census Data	cfincgrp	Median Census Family Income Groupings (Median Census Incomes Were Derived For 17 Public Health Service Areas within Capital Health). Each woman was initially assigned the income value for the PHSA then the income was grouped and values 0-3 were assigned.	0= $60-69000 1= $50-59999. 2= $40-49999 3= $30-39999
lessgr9	Census Data	gr9grp	Less Than Grade 9 Education Groupings (% of less than grade 9 education for the 17 PHS areas were grouped into 3 values)	0= 2-5% (Low Risk) 1=6-9% (Moderate Risk) 2=11-16% (High Risk)
bacdeg	Census Data	bacdeggp	Baccalaureate degree or greater. Education Groupings (% baccalaureate degree or greater. Education for the 17 PHS areas were grouped into 3 values)	0= 21,26,40% (Low Risk) 1=11,12,13,16, 17% (Moderate Risk) 2= 5-10% (High Risk)
sparent	Census Data	spargrp	Single Parent Grouping (% Single Parent for the 17 PHS areas were grouped into 3 values).	0= 6,8,9,10% (Low Risk) 1=11,15,.16, 7,18% (Moderate Risk) 2=20, 24% (High Risk)
aborigin	Census Data	aborgrp	Aboriginal Population Grouping (%Aboriginal Population for the 17 PHS areas were grouped into 3 values).	0= 1.1, 1.2, 2.0% (Low Risk) 1=2.3, 2.6, 2.7% (Moderate Risk) 2=5.1,5.7,6.3. 9.8 % (High Risk)

Data Transformations and Data Combinations

Variable Original Data Assembled Variable Name)	Data Source	Variable Name	Label Description	Values
Socio-Economic Factor Analysis				
chainc, sparent aborigin, lessgr9 bacdeg	Census Data	fac1_1	REGR Factor Score	-1.26310 -1.22594 -.97419 -.90090 -.77537 -.65709 -.47547 -.31089 .07244 .10615 .15708 .16952 .44461 .85352 1.10713 1.19599 2.47650 missing data (1387 records)
chainc sparent aborigin, lessgr9 bacdeg	Census Data	fac2_2**	REGR Factor Score	-1.61562 -1.28556 -1.04410 -.48670 -.33795 -.33375 -.32247 -.28439 -.12041 .00974 .05477 .12614 .42016 .71095 .76407 .95404 2.79108 missing data (1387 records)

Appendix D: Calculation and Interpretation of Geographic Rates for Public Health Service Areas

Calculation and Interpretation of Geographic Rates for Public Health Service Areas

Within the study, the distribution of maternal risk factors and the distribution of low birth weight infants and preterm infants has been presented according to the PHSA that the mother resides in. The information has been presented in the form of a colored map and a graph. The method for calculation of the risk factor and birth outcome rates consists of several steps:

1.Calculate the rates for each PHSA.

Eg:

PHSA	Number of Low Birth Weight Infants	Total # Live Births	Proportion Low Birth Weight
Central	123	1,809	0.0679

2. Calculate the rate for the Capital Health Region.

Number of Low Birth Weight Infants: 1,269
Total Number of Live Births: 26,265
Proportion Low Birth Weight 1,269/ 26,265=0.048

3. Calculate the standard error of a probability of the risk factor or health event for each PHSA using the following formula: square root of p (1-p)/n where p is the proportion (estimate of probability) for the area and n is the number of births.

Eg:

PHSA	Number of LBW	Total # Live Births	Proportion LBW	Calculation	Standard Error
Central	123	1,809	0.0679	$\sqrt{\frac{0.06\,(1-0.06)}{1809}}$	0.005

Calculation and Interpretation of Geographic Rates for Public Health Service Areas (cont'd)

4. Calculate the PHSA specific standard scores.

This is achieved by subtracting the Capital Health Regional proportion from the PHSA proportion and dividing these by the standard error derived for each PHSA in step 3. This is completed for every PHSA.

$$\frac{\text{PHSA proportion- Capital Health Region Proportion}}{\text{PHSA standard error}}$$

5. Graph the standard scores calculated in step 4.

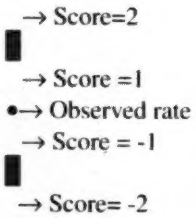
To facilitate the interpretation of the PHSA standard scores, the following color scheme was used to differentiate the PHSA rates that may differ from the Capital Health Region.

Score	Interpretation	Color in Map
>2	Higher than the Capital Health Regional Average (significant differences in a conventional statistical test (p<.05))	Red
1 to 2	Probably higher than Capital Health Regional Average (p>0.5 but < 0.95 that difference is not due to random variation)	Orange
1 to-1	Not likely to differ from the Capital Health Regional Average (p>.05 that difference is not due to random variation)	Yellow
-1 to -2	Probably lower than Capital Health Regional Average (p>0.5 but < 0.95 that difference is not due to random variation)	Light Green
<-2	Lower than Capital Health Regional Average (significant difference in a conventional statistical test (p<0.05))	Dark Green

Calculation and Interpretation of Geographic Rates for Public Health Service Areas (cont'd)

Calculation and Interpretation of Geographic Rates for Public Health Service Areas (cont'd)

The black dot represents the value of the observed rate for each Public Health Service Area. The colors of the bars above and below the dot correspond to the score of the PHSA. The portion of the bar closest to the black dot represents the value for a standard score of 1-1, while the part of the bars farthest from the dot represent the value for a score of 2 or –2.



6. Generate maps using the same categories for each PHSA as listed in step 5

The graph and map are placed on the same page. The map provides a quick overview while more detailed information is presented in the graph. The color assigned to each PHSA is based on the color of the bars in the graph for the same PHSA. This provides a spatial context to the distribution patterns and consistency amongst the two graphic components.

Appendix E: Adjusted Odds Ratios for the General Model of Maternal Risk Factors and Preterm Birth

Risk Factor	**16**	**15**	**14**	**13**	**12**	**11**	**10**	**9**	**8**	**7**
Age Younger Age^1 Older Age^2	0.89^1 (0.72-1.10) 1.16^2 (0.99-1.36)	0.87^1 (0.71-1.08) *1.17^2 (1.00-1.37)	0.87^1 (0.71-1.07) *1.19^2 (1.02-1.39)	0.85^1 (0.69-1.05) *1.19^2 (1.02-1.39)	0.85^1 (0.69-1.05) *1.20^2 (1.03-1.40)	0.82^1 (0.69-1.00) *1.25^2 (1.08-1.45)	*0.81^1 (0.66-0.99) *1.26^2 (1.08-1.45)	0.81^1 (0.67-1.00) *1.28^2 (1.10-1.48)	0.82^1 (0.67-1.00) *1.28^2 (1.10-1.47)	*0.79 (0.65-0.97) *1.30 (1.13-1.50)
Genetic Ht < 152 cm Wt ≥ 91kg^1 Wt ≤ 45kg^2	1.05 (0.76-1.44) 0.89^1 (0.72-1.10) 0 .80^2 (0.35-1.87)	1.06 (0.77-1.46) 0.88^1 (0.71-1.08) 0.79^2 (0.34-1.84)	1.10 (0.81-1.50) 0.87^1 (0.71-1.07) 0.78^2 (0.34-1.79)	1.08 (0.79-1.48) 0.88^1 (0.72-1.08) 0 .77^2 (0.34-1.77)	1.10 (0.81-1.50) 0.87^1 (0.71-1.06) 0.76^2 (0.34-1.71)	1.11 (0.82-1.52) 0.87^1 (0.71-1.07) 0.78^2 (0.35-1.74)	1.09 (0.80-1.48) 0.96^1 (0.79-1.18) 0 .86^2 (0.39-1.89)	1.11 (0.82-1.50) 0.98^1 (0.80-1.20) 0.88^2 (0.41-1.89)	1.11 (0.82-1.50) 0.98^1 (0.80-1.20) 0.89^2 (0.41-1.92)	1.10 (0.81-1.48) 1.06^1 (0.88-1.30) 0.87^2 (0.40-1.87
Obstetrical History Abortion	1.11 (0.99-1.24)	1.11 (0.99-1.24)	*1.11 (1.00-1.24)	*1.13 (1.01-1.26)	*1.13- (1.01-1.26)	*1.13 (1.01-1.26)	*1.13 (1.01-1.26)	*1.15 (1.03-1.28)	*1.15 (1.03-1.28)	*1.16 (1.04-1.29)
Preterm Birth	*4.33 (3.58-5.23)	*4.29 (3.55-5.19)	*4.32 (3.59-5.21)	*4.31 (3.58-5.19)	*4.36 (3.62-5.24)	*4.34 (3.61-5.21)	*4.35 (3.62-5.23)	*4.39 (3.66-5.26)	*4.39 (3.66-5.26)	*4.43 (3.69-5.31)
Nulliparity	*1.66 (1.24-2.22)	*1.64 (1.22-2.19)	*1.58 (1.19-2.11)	*1.58 (1.18-2.10)	*1.60 (1.20-2.13)	*1.59 (1.19-2.12)	*1.66 (1.25-2.21)	*1.73 (1.30-2.30)	*1.73 (1.30-2.30)	*1.72 (1.30-2.28)
Stillbirth	0.85 (0.58-1.23)	0.85 (0.59-1.24)	0.90 (0.63-1.30)	0.90 (0.62-1.29)	0.91 (0.63-1.30)	0.91 (0.63-1.30)	0.89 (0.62-1.28)	0.93 (0.65-1.34)	0.94 (0.65-1.34)	0.96 (0.67-1.37)
Neonatal Death	1.06 (0.67-1.68)	1.06 (0.67-1.68)	1.14 (0.73-1.78)	1.15 (0.74-1.79)	1.15 (0.74-1.79)	1.14 (0.73-1.79)	1.15 (0.74-1.79)	1.16 (0.75-1.80)	1.16 (0.75-1.80)	1.13 (0.73-1.76)
SGA Birth	1.37 (0.81-2.31)	1.36 (0.81-2.28)	1.38 (0.83-2.28)	1.39 (0.84-2.30)	1.37 (0.83-2.27)	1.37 (0.84-2.29)	1.38 (0.84-2.29)	1.56 (0.95-2.56)	1.57 (0.96-2.58)	1.59 (0.97-2.62)
Prenatal Care Missing Visits Data	0.97 (0.83-1.14)	0.97 (0.82-1.13)	0.95 (0.82-1.12)	0.95 (0.81-1.11)	0.94 (0.80-1.10)	0.94 (0.80-1.10)	0.94 (0.80-1.10)	0.93 (0.80-1.09)	0.93 (0.80-1.09)	0.93 (0.80-1.08)
≤ 4 Visits	*4.61 (4.00-5.30)	*4.64 (4.04-5.34)	*4.79 (4.17-5.49)	*4.82 (4.20-5.53)	*4.85 (4.23-5.56)	*4.84 (4.22-5.55)	*4.83 (4.22-5.54)	*4.82 (4.22-5.52)	*4.82 (4.22-5.52)	*4.84 (4.23-5.53)
5-7 Visits	*4.05 (3.46-4.74)	*4.07 (3.48-4.76)	*4.14 (3.55-4.82)	*4.11 (3.53-4.80)	*4.15 (3.56-4.84)	*4.13 (3.54-4.81)	*4.11 (3.53-4.79)	*4.06 (3.49-4.72)	*4.06 (3.49-4.72)	*4.06 (3.49-4.73)
Missing Classes Data	1.09 (0.92-1.28)	1.09 (0.92-1.28)	1.06 (0.90-1.25)	1.06 (0.90-1.25)	1.07 (0.91-1.25)	1.07 (0.91-1.25)	1.08 (0.92-1.27)	1.08 (0.92-1.27)	1.08 (0.92-1.27)	1.07 (0.92-1.26)
No Classes	*1.49 (1.13-1.96)	*1.49 (1.13-1.96)	*1.47 (1.12-1.92)	*1.46 (1.11-1.91)	*1.47 (1.12-1.93)	*1.46 (1.12-1.92)	*1.44 (1.10-1.89)	*1.47 (1.12-1.92)	*1.47 (1.12-1.92)	*1.44 (1.10-1.89)
Classes x Parity Interaction	1.06 (0.77-1.46)	1.07 (0.78-1.47)	1.13 (0.83-1.54)	1.13 (0.83-1.55)	1.14 (0.83-1.55)	1.14 (0.83-1.55)	1.15 (0.84-1.56)	1.12 (0.82-1.52)	1.12 (0.82-1.52)	1.16 (0.85-1.57)
Lifestyle Smokers	0.97 (0.85-1.10)	0.98 (0.86-1.11)	0.99 (0.88-1.13)	0.99 (0.87-1.12)	0.99 (0.87-1.12)	1.00 (0.88-1.13)	0.97 (0.86-1.10)	0.98 (0.86-1.10)	0.98 (0.86-1.10)	0.98 (0.87-1.11)
Quitters	0.96 (0.68-1.36)	0.96 (0.68-1.36)	1.00 (0.72-1.41)	1.01 (0.72-1.41)	1.03 (0.73-1.44)	1.02 (0.73-1.43)	1.04 (0.74-1.45)	1.04 (0.75-1.45)	1.04 (0.75-1.46)	1.05 (0.75-1.46)
Alcohol Use	0.61 (0.36-1.04)	0.62 (0.37-1.05)	0.62 (0.37-1.04)	0.63 (0.38-1.06)	0.64 (0.38-1.07)	0.64 (0.38-1.07)	0.63 (0.38-1.05)	0.63 (0.38-1.04)	0.63 (0.38-1.04)	0.63 (0.38-1.04)
Street Drug Use	*1.36 (1.00-1.84)	1.34 (0.99-1.82)	*1.38 (1.02-1.86)	*1.36 (1.01-1.83)	*1.37 (1.01-1.84)	*1.37 (1.02-1.84)	*1.38 (1.02-1.85)	*1.40 (1.04-1.87)	*1.40 (1.05-1.87)	*1.40 (1.05-1.88)
Missing Street Drug Use Data	1.08 (0.74-1.57)	1.11 (0.76-1.61)	1.09 (0.76-1.57)	1.09 (0.76-1.58)	1.09 (0.76-1.57)	1.10 (0.76-1.58)	1.09 (0.76-1.57)	1.10 (0.77-1.57)	1.10 (0.77-1.57)	1.08 (0.76-1.55)
Alcohol x Smoking Interaction	*2.14 (1.18-3.85)	*2.08 (1.16-3.72)	*2.05 (1.15-3.65)	*2.05 (1.15-3.63)	*2.01 (1.13-3.56)	*2.00 (1.13-3.55)	*2.02 (1.14-3.56)	*2.05 (1.16-3.61)	*2.06 (1.17-3.63)	*2.02 (1.15-3.54)
Pre-existing Disease Heart Disease	1.56 (0.86-2.83)	1.53 (0.84-2.79)	1.55 (0.86-2.79)	1.56 (0.87-2.81)	1.53 (0.85-2.76)	1.53 (0.85-2.75)	1.53 (0.85-2.74)	1.49 (0.83-2.68)	1.48 (0.82-2.67)	
Diabetes Mellitus	*2.13 (1.42-3.19)	*2.12 (1.41-3.17)	*1.99 (1.33-2.98)	*1.96 (1.31-2.93)	*1.98 (1.33-2.95)	*2.00 (1.35-2.98)	*2.00 (1.35-2.96)	*2.29 (1.57-3.34)	*2.30 (1.58-3.35)	
Chronic Hypertension	*1.68 (1.12-2.53)	*1.67 (1.11-2.52)	*1.61 (1.08-2.42)	*1.65 (1.10-2.47)	*1.65 (1.11-2.44)	*1.67 (1.13-2.47)	*2.53 (1.74-3.67)	*2.67 (1.85-3.85)	*2.67 (1.85-3.85)	
Renal Disease	*4.14 (1.62-10.54)	*4.11 (1.61-10.49)	*4.12 (1.64-10.36)	*4.02 (1.62-10.00)	*4.65 (1.93-11.17)	*4.79 (2.01-11.40)	*5.36 (2.29-12.56)	*5.81 (2.48-13.60)	*5.89 (2.52-13.77)	
Other Medical Diseases	1.26 (0.98 -1.61)	1.24 (0.97-1.59)	1.21 (0.96-1.56)	1.23 (0.96-1.57)	1.24 (0.97-1.58)	1.24 (0.97-1.58)	1.25 (0.98-1.59)	1.26 (0.99-1.60)	1.26 (0.99-1.61)	
Nutrition Anemia	1.17 (0.57-2.40)	1.21 (0.59-2.47)	1.21 (0.61-2.43)	1.27 (0.63-2.52)	1.26 (0.63-2.51)	1.25 (0.62-2.48)	1.25 (0.63-2.49)	1.23 (0.62-2.45)		
Poor Gestational Weight Gain	1.03 (0.65-1.63)	1.01 (0.64-1.61)	1.02 (0.65-1.61)	1.02 (0.65-1.61)	1.02 (0.65-1.60)	1.02 (0.65-1.60)	1.05 (0.67-1.65)	1.18 (0.75-1.84)		
Problems in Current Pregnancy Acute Medical Problems	*2.98 (1.70-5.19)	*3.03 (1.74-5.28)	*3.18 (1.86-5.43)	*3.13 (1.83-5.36)	*3.14 (1.83-5.37)	*3.14 (1.83-5.37)	*3.00 (1.75-5.14)			
Poly/Oligo Hydramnios	*4.03 (3.21-5.06)	*3.99 (3.18-5.01)	*4.15 (3.33-5.17)	*4.23 (3.40-5.30)	*4.19 (3.37-5.22)	*4.19 (3.37-5.21)	*4.17 (3.36-5.18)			
Gestational Diabetes	1.29 (0.99-1.67)	1.27 (0.98-1.65)	1.28 (0.99-1.66)	1.28 (0.99-1.60)	1.26 (0.97-1.63)	1.26 (0.98-1.63)	*1.33 (1.04-1.72)			
Complications Gestational Hypertension	*1.86 (1.40-2.46)	*1.86 (1.40-2.47)	*1.77 (1.33-2.35)	*1.74 (1.31-2.30)	*2.74 (2.19-3.43)	*2.86 (2.35-3.49)				
Gest Hyp x Age Interaction Younger^1 Older^2	*0.35^1 (0.14-0.87) 1.68^2 (0.99-2.84)	*0.35^1 (0.14-0.88) 1.67^2 (0.99-2.83)	*0.33^1 (0.13-0.84) 1.64^2 (0.97-2.75)	*0.34^1 (0.14-0.86) 1.62^2 (0.97-2.73)	0.50^1 (0.21-1.20) *1.68^2 (1.02-2.76)					
Pre-eclampsia	*3.60 (2.45-5.30)	*3.60 (2.45-5.30)	*3.61 (2.45-5.30)	*3.59 (2.44-5.26)						
Bleeding < 20weeks	*1.75 (1.40-2.18)	*1.81 (1.45-2.25)	*2.26 (1.84-2.78)							
Bleeding ≥ 20 weeks	*5.05 (4.15-6.13)	*5.80 (4.81-7.00)								
Placenta Previa	*6.97 (3.98-12.19)									

*Significant $p < 0.05$

	9	8	7	6	5	4	3	2	1
	0.81[1] (0.67-1.00) *1.28[2] (1.10-1.48)	0.82[1] (0.67-1.00) *1.28[2] (1.10-1.47)	*0.79 (0.65-0.97) *1.30 (1.13-1.50)	*0.79[1] (0.65-0.97) *1.30[2] (1.12-1.50)	0.83[1] (0.68-1.01) *1.28[2] (1.11-.1.48)	0.83[1] (0.68-1.01) *1.28[2] (1.11-1.48)	*1.23[1] (1.02-1.49) *1.18[2] (1.03-1.36)	*1.29[1] (1.07-1.55) *1.19[2] (1.04-1.36)	*1.29[1] (1.07-1.55) *1.19[2] (1.04-1.37)
	1.11 (0.82-1.50) 0.98[1] (0.80-1.20) 0.88[2] (0.41-1.89)	1.11 (0.82-1.50) 0.98[1] (0.80-1.20) 0.89[2] (0.41-1.92)	1.10 (0.81-1.48) 1.06[1] (0.88-1.30) 0.87[2] (0.40-1.87	1.10 (0.81-1.49) 1.06[1] (0.87-1.29) 0.87[2] (0.41-1.88)	1.08 (0.84-1.46) 1.06[1] (0.87-1.28) 0.88[2] (0.41-1.89)	1.08 (0.80-1.47) 1.05 (0.87-1.28) 0.88[2] (0.41-1.90)	1.20 (0.89-1.61) 0.97[1] (0.80-1.17) 0.95[2] (0.45-2.00)	1.24 (0.93-1.66) 0.98[1] (0.81-1.18) 1.05[2] (0.51-2.18)	
	*1.15 (1.03-1.28)	*1.15 (1.03-1.28)	*1.16 (1.04-1.29)	*1.16 (1.04-1.29)	*1.18 (1.06-1.31)	*1.18 (1.06-1.31)	*1.19 (1.07-1.33)		
	*4.39 (3.66-5.26)	*4.39 (3.66-5.26)	*4.43 (3.69-5.31)	*4.44 (3.71-5.32)	*4.49 (3.75-5.37)	*4.49 (3.75-5.37)	*4.65 (3.91-5.53)		
	*1.73 (1.30-2.30)	*1.73 (1.30-2.30)	*1.72 (1.30-2.28)	*1.73 (1.30-2.30)	*1.73 (1.30-2.29)	*1.95 (1.73-2.19)	*1.39 (1.25-1.54)		
	0.93 (0.65-1.34)	0.94 (0.65-1.34)	0.96 (0.67-1.37)	0.96 (0.67-1.37)	0.96 (0.67-1.38)	0.96 (0.68-1.38)	1.00 (0.74-1.42)		
	1.16 (0.75-1.80)	1.16 (0.75-1.80)	1.13 (0.73-1.76)	1.13 (0.73-1.75)	1.13 (0.73-1.75)	1.14 (0.73-1.76)	1.23 (0.81-1.87)		
	1.56 (0.95-2.56)	1.57 (0.96-2.58)	1.59 (0.97-2.62)	1.59 (0.97-2.62)	1.60 (0.98-2.62)	1.60 (0.97-2.62)	*1.80 (1.12-2.88)		
	0.93 (0.80-1.09)	0.93 (0.80-1.09)	0.93 (0.80-1.08)	0.93 (0.79-1.08)	0.92 (0.79-1.07)	0.92 (0.79-1.07)			
	*4.82 (4.22-5.52)	*4.82 (4.22-5.52)	*4.84 (4.23-5.53)	*4.84 (4.24-5.54)	*5.03 (4.41-5.75)	*5.03 (4.40-5.74)			
	*4.06 (3.49-4.72)	*4.06 (3.49-4.72)	*4.06 (3.49-4.73)	*4.07 (3.50-4.73)	*4.09 (3.52-4.76)	*4.09 (3.52-4.76)			
	1.08 (0.92-1.27)	1.08 (0.92-1.27)	1.07 (0.92-1.26)	1.07 (0.92-1.26)	*1.18 (1.02-1.37)	*1.19 (1.03-1.37)			
	*1.47 (1.12-1.92)	*1.47 (1.12-1.92)	*1.44 (1.10-1.89)	*1.45 (1.11-1.90)	*1.47 (1.13-1.93)	*1.64 (1.43-1.88)			
	1.12 (0.82-1.52)	1.12 (0.82-1.52)	1.16 (0.85-1.57)	1.15 (0.851-1.56)	1.15 (0.84-1.56)				
	0.98 (0.86-1.10)	0.98 (0.86-1.10)	0.98 (0.87-1.11)	1.01 (0.90-1.14)					
	1.04 (0.75-1.45)	1.04 (0.75-1.46)	1.05 (0.75-1.46)	1.04 (0.74-1.44)					
	0.63 (0.38-1.04)	0.63 (0.38-1.04)	0.63 (0.38-1.04)	1.07 (0.85-1.34)					
	*1.40 (1.04-1.87)	*1.40 (1.05-1.87)	*1.40 (1.05-1.88)	*1.49 (1.12-1.99)					
	1.10 (0.77-1.57)	1.10 (0.77-1.57)	1.08 (0.76-1.55)	1.02 (0.72-1.45)					
	*2.05 (1.16-3.61)	*2.06 (1.17-3.63)	*2.02 (1.15-3.54)						
	1.49 (0.83-2.68)	1.48 (0.82-2.67)							
	*2.29 (1.57-3.34)	*2.30 (1.58-3.35)							
	*2.67 (1.85-3.85)	*2.67 (1.85-3.85)							
	*5.81 (2.48-13.60)	*5.89 (2.52-13.77)							
	1.26 (0.99-1.60)	1.26 (0.99-1.61)							
	1.23 (0.62-2.45)								
	1.18 (0.75-1.84)								

Appendix F: Adjusted Odds Ratios for the Model of Maternal Risk Factors, Socio-economic Status and Preterm Birth

Risk Factor	**17**	**16**	**15**	**14**	**13**	**12**	**11**	**10**	**9**	**8**
Age Younger Age^1 Older Age^2	0.89^1 (0.70-1.11) 1.14^2 (0.97-1.30)	0.87^1 (0.69-1.10) 1.17^2 (0.98-1.37)	0.87^1 (0.69-1.09) *1.17^2 (1.00-1.37)	0.85^1 (0.68-1.06) *1.18^2 (1.08-1.38)	0.84^1 (0.67-1.06) * 1.18^2 (1.01-1.38)	0.83^1 (0.66-1.03) *1.24^2 (1.07-1.44)	0.82^1 (0.66-1.02) *1.24^2 (1.07-1.44)	0.82^1 (0.66-1.02) *1.27^2 (1.09-1.47)	0.82^1 (0.66-1.02) *1.27^2 (1.09-1.47)	*0.80 (0.66-.0.99) *1.29 (1.11-1.50)
SES Fac1_1	0.96 (0.91-1.01)	0.96 (0.91-1.01)	0.96 (0.91-1.01)	0.96 (0.91-1.01)	0.96 (0.91-1.01)	0.96 (0.91-1.01)	0.95 (0.90-1.01)	0.95 (0.90-1.01)	0.95 (0.90-1.01)	0.96 (0.91-1.01)
Fac2_2	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.04 (0.98-1.12)	1.04 (0.98- 1.11)	1.04 (0.97-1.11)	1.05 (0.98-1.12)
Genetic Ht < 152 cm Wt ≥ 91kg^1 Wt ≤ 45kg^2	1.06 (0.77-1.50) 0.88^1 (0.71-1.10) 0.92^2 (0.39-2.20)	1.08 (0.78-1.49) 0.87^1 (0.70-1.08) 0.90^2 (0.38-2.12)	1.12 (0.82-1.55) 0.86^1 (0.69-1.07) 0.90^2 (0.39-2.07)	1.11 (0.81-1.53) 0.87^1 (0.70-1.08) 0.87^2 (0.38-2.00)	1.12 (0.82-1.55) 0.86^1 (0.69-1.06) 0.87^2 (0.38-1.95)	1.13 (0.82-1.56) 0.86^1 (0.69-1.12) 0.88^2 (0.39-1.97)	1.10 (0.80-1.52) 0.96^1 (0.77-1.18) 0.95^2 (0.43-2.10)	1.12 (0.82-1.53) 0.98^1 (0.79-1.20) 0.98^2 (0.45-2.10)	1.12 (0.82-1.54) 0.98^1 (0.79-1.20) 0.98^2 (0.45-2.11)	1.10 (0.81-1.51) 1.05^1 (0.86-1.30) 0.95^2 (0.44-2.05)
Obstetrical History Abortion	1.11 (0.98-1.25)	1.11 (0.98-1.25)	*1.12 (1.00-1.26)	*1.13 (1.01-1.27)	*1.14 (1.01-1.28)	*1.14 (1.01-1.28)	*1.14 (1.01-1.27)	*1.15 (1.03-1.29)	*1.15 (1.03-1.29)	*1.16 (1.04-1.30)
Preterm Birth	*4.55 (3.73-5.51)	*4.51 (3.70-5.49)	*4.54 (3.74-5.50)	*4.53 (3.74-5.49)	*4.57 (3.77-5.54)	*4.55 (3.75-5.51)	*4.57 (3.78-5.54)	*4.56 (3.77-5.51)	*4.56 (3.77-5.51)	*4.58 (3.80-5.54)
Nulliparity	*1.59 (1.18-2.13)	*1.57 (1.17-2.11)	*1.52 (1.13-2.03)	*1.51 (1.13-2.02)	*1.55 (1.15-2.05)	*1.53 (1.14-2.04)	*1.60 (1.20-2.13)	*1.67 (1.25-2.22)	*1.67 (1.25-2.22)	*1.66 (1.25-2.21)
Stillbirth	0.91 (0.62-1.31)	0.92 (0.62-1.35)	0.98 (0.67-1.43)	0.97 (0.66-1.40)	0.98 (0.67-1.42)	0.98 (0.67-1.42)	0.96 (0.66-1.39)	1.00 (0.69-1.45)	1.00 (0.70-1.45)	1.03 (0.72-1.48)
Neonatal Death	0.94 (0.58-1.51)	0.94 (0.58-1.52)	1.01 (0.63-1.62)	1.02 (0.64-1.63)	1.02 (0.64-1.63)	1.01 (0.63-1.62)	1.03 (0.64-1.64)	1.05 (0.66-1.68)	1.05 (0.66-1.67)	1.03 (0.65-1.64)
SGA Birth	1.52 (0.91-2.53)	1.51 (0.89-2.54)	1.54 (0.93-2.55)	1.55 (0.93-2.56)	1.53 (0.92-2.54)	1.52 (0.91-2.53)	1.55 (0.93-2.56)	*1.75 (1.06-2.88)	*1.76 (1.07-2.89)	*1.79 (1.08-2.94)
Prenatal Care Missing Visits Data	0.99 (0.84-1.20)	0.98 (0.83-1.17)	0.97 (0.83-1.15)	0.96 (0.82-1.14)	0.95 (0.81-1.13)	0.95 (0.81-1.12)	0.95 (0.81-1.12)	0.95 (0.80-1.11)	0.95 (0.80-1.11)	0.94 (0.80-1.11)
≤ 4 Visits	*4.45 (3.84-5.20)	*4.50 (3.89-5.21)	*4.63 (4.01-5.35)	*4.68 (4.05-5.40)	*4.70 (4.07-5.42)	*4.69 (4.07-5.42)	*4.70 (4.07-5.42)	*4.71 (4.09-5.42)	*4.71 (4.09-5.42)	*4.71 (4.09-5.42)
5-7 Visits	*4.27 (3.63-5.01)	*4.29 (3.66-5.04)	*4.35 (3.71-5.09)	*4.31 (3.68-5.05)	*4.34 (3.71-5.08)	*4.33 (3.70-5.07)	*4.30 (3.68-5.03)	*4.24 (3.63-4.95)	*4.24 (3.63-4.95)	*4.24 (3.63-4.95)
Missing Classes Data	1.06 (0.89-1.25)	1.06 (0.89-1.25)	1.03 (0.87-1.22)	1.03 (0.87-1.22)	1.03 (0.87-1.22)	1.03 (0.87-1.22)	1.04 (0.88-1.23)	1.04 (0.88-1.23)	1.04 (0.88-1.23)	1.04 (0.88-1.22)
No Classes	*1.41 (1.07-1.87)	*1.42 (1.07-1.88)	*1.40 (1.06-1.84)	*1.39 (1.05-1.83)	*1.40 (1.06-1.84)	*1.39 (1.06-1.83)	*1.38 (1.05-1.82)	*1.40 (1.07-1.84)	*1.40 (1.07-1.84)	*1.38 (1.05-1.81)
Classes x Parity Interaction	1.15 (0.83-1.60)	1.16 (0.84-1.60)	1.22 (0.89-1.68)	1.23 (0.89-1.69)	1.23 (0.90-1.69)	1.23 (0.90-1.69)	1.24 (0.91-1.70)	1.21 (0.89-1.66)	1.21 (0.89-1.66)	1.24 (0.91-1.70)
Lifestyle Smokers	0.97 (0.85-1.10)	0.98 (0.86-1.12)	1.00 (0.87-1.13)	0.99 (0.87-1.13)	0.99 (0.87-1.13)	0.99 (0.87-1.13)	0.97 (0.86-1.11)	0.98 (0.86-1.12)	0.98 (0.86-1.12)	0.98 (0.86-1.12)
Quitters	0.95 (0.65-1.40)	0.94 (0.65-1.36)	0.99 (0.70-1.42)	1.00 (0.70-1.42)	1.02 (0.72-1.45)	1.02 (0.72-1.45)	1.03 (0.72-1.46)	1.01 (0.71-1.43)	1.01 (0.71-1.43)	1.02 (0.72-1.44)
Alcohol Use	0.68 (0.40-1.10)	0.69 (0.41-1.15)	0.69 (0.41-1.15)	0.70 (0.42-1.17)	0.70 (0.42-1.17)	0.70 (0.42-1.17)	0.69 (0.41-1.15)	0.69 (0.41-1.14)	0.69 (0.41-1.14)	0.67 (0.44-1.11)
Street Drug Use	1.21 (0.85-1.70)	1.20 (0.85-1.71)	1.25 (0.89-1.79)	1.25 (0.89-1.76)	1.25 (0.89-1.76)	1.26 (0.89-1.77)	1.25 (0.89-1.76)	1.25 (0.89-1.76)	1.25 (0.89-1.76)	1.25 (0.90-1.75)
Missing Street Drug Use Data	1.19 (0.78-1.79)	1.23 (0.81-1.86)	1.19 (0.79-1.79)	1.18 (0.78-1.77)	1.17 (0.78-1.76)	1.17 (0.78-1.76)	1.18 (0.79-1.77)	1.21 (0.81-1.76)	1.21 (0.81-1.80)	1.19 (0.80-1.78)
Alcohol x Smoking Interaction	*1.93 (1.06-3.49)	*1.87 (1/04-3.39)	*1.87 (1.05-3.36)	*1.87 (1.05-3.33)	*1.85 (1.04-3.31)	*1.85 (1.03-3.30)	*1.88 (1.05-3.36)	*1.87 (1.05-3.33)	*1.88 (1.06-3.34)	*1.89 (1.06-3.35)
Pre-existing Disease Heart Disease	1.62 (0.87-3.00)	1.59 (0.85-2.97)	1.63 (0.88-3.00)	1.64 (0.89-3.02)	1.60 (0.87-2.96)	1.59 (0.86-2.95)	1.60 (0.87-2.95)	1.58 (0.85-2.91)	1.57 (0.85-2.90)	
Diabetes Mellitus	*2.19 (1.45-3.30)	*2.18 (1.45-3.29)	*2.05 (1.37-3.08)	*2.03 (1.35-3.04)	*2.03 (1.36-3.04)	*2.06 (1.38-3.07)	*2.04 (1.37-3.04)	*2.35 (1.60-3.45)	*2.36 (1.61-3.45)	
Chronic Hypertension	*1.60 (1.04-2.4)	*1.58 (1.03-2.43)	*1.53 (1.00-2.35)	*1.58 (1.04-2.41)	*1.59 (1.05-2.39)	*1.60 (1.06-2.42)	*2.42 (1.64-3.57)	*2.59 (1.76-3.79)	*2.58 (1.76 -3.79)	
Renal Disease	*4.61 (1.67-13.00)	*4.58 (1.65-12.69)	*4.64 (1.70-12.60)	*4.52 (1.68-12.20)	*5.09 (1.96-13.21)	*5.25 (2.05-13.41)	*5.83 (2.33-14.50)	*6.57 (2.63-16.40)	*6.64 (2.66-16.50)	
Other Medical Diseases	*1.29 (1.00-1.70)	1.28 (0.99-1.65)	1.25 (0.97-1.61)	1.25 (0.97-1.61)	1.26 (0.98-1.62)	1.26 (0.98-1.62)	1.27 (0.98-1.63)	1.27 (0.99-1.63)	1.27 (0.99-1.63)	
Nutrition Anemia	1.16 (0.52-2.50)	1.20 (0.54-2.66)	1.18 (0.55-2.54)	1.25 (0.59-2.66)	1.25 (0.59-2.66)	1.24 (0.58-2.64)	1.26 (0.59-2.67)	1.26 (0.59-2.66)		
Poor Gestational Weight Gain	0.99 (0.60-1.60)	0.97 (.595-1.59)	0.98 (0.60-1.60)	0.97 (0.59-1.58)	0.96 (0.59-1.57)	0.96 (0.59-1.57)	0.98 (0.60-1.60)	1.10 (0.68-1.78)		
Problems in Current Pregnancy Acute Medical Problems	*3.33 (1.90-5.80)	*3.39 (1.94-5.94)	*3.56 (2.07-6.11)	*3.51 (2.04-6.05)	*3.52 (2.05-6.06)	*3.52 (2.05-6.06)	*3.37 (1.95-5.81)			
Poly/Oligo hydramnios	*4.09 (3.22-5.20)	*4.05 (3.19-5.13)	*4.21 (3.34-5.29)	*4.28 (3.41-5.38)	*4.25 (3.39-5.34)	*4.24 (3.37-5.32)	*4.22 (3.37-5.29)			
Gestational Diabetes	*1.34 (1.02-1.72)	*1.31 (1.01-1.72)	*1.32 (1.02-1.72)	*1.34 (1.02-1.72)	*1.31 (1.01-1.69)	*1.31 (1.01-1.69)	*1.39 (1.08-1.80)			
Complications Gestational Hypertension	*1.82 (1.35-2.44)	*1.82 (1.35-2.44)	*1.71 (1.28-2.30)	*1.68 (1.25-2.26)	*2.66 (2.10-3.35)	*2.86 (2.33-3.52)				
Gest Hyp x Age Interaction Younger^1 Older^2	0.54^1 (0.21-1.40) *1.78^2 (1.03-3.06)	0.54^1 (0.21-1.39) *1.78^2 (1.03-3.06)	0.53^1 (0.20-1.36) *1.76^2 (1.02-3.01)	0.54^1 (0.21-1.38) *1.74^2 (1.02-2.97)	0.71^1 (0.29-1.75) *1.77^2 (1.06-2.96)					
Pre-eclampsia	*3.53 (2.37-5.26)	*3.53 (2.37-5.26)	*3.57 (2.40-5.31)	*3.55 (2.39-5.30)						
Bleeding < 20 weeks	*1.80 (1.44-2.30)	*1.86 (1.49-2.32)	*2.33 (1.89-2.89)							
Bleeding ≥ 20 weeks	*5.05 (4.12-6.20)	*5.86 (4.82-7.12)								
Placenta Previa	*6.71 (3.81-11.80)									

*Significant $p < 0.05$

10	9	8	7	6	5	4	3	2	1
0.82^{1} .66-1.02) $*1.27^{2}$ .09-1.47)	0.82^{1} (0.66-1.02) $*1.27^{2}$ (1.09-1.47)	*0.80 (0.66-.0.99) *1.29 (1.11-1.50)	*0.79 (0.64-0.99) *1.29 (1.11-1.50)	0.82^{1} (0.66-1.01) $*1.29^{2}$ (1.11-1.49)	0.83^{1} (0.67-1.10) $*1.29^{2}$ (1.14-.1.49)	1.15^{1} (0.94-1.41) $*1.22^{2}$ (1.05-1.40)	1.21^{1} (0.99-1.48) $*1.23^{2}$ (1.06-1.41)	1.21^{1} (0.99-1.47) $*1.23^{2}$ (1.07-1.42)	$*1.25^{1}$ (1.02-1.5) $*1.22^{2}$ (1.06-1.40)
0.95 .90-1.01)	0.95 (0.90-1.01)	0.96 (0.91-1.01)	0.96 (0.92-1.01)	0.97 (0.92-1.05)	0.97 (0.92-1.02)	*1.07 (1.02-1.13)	*1.09 (1.04-1.15)	*1.09 (1.04-1.15)	
1.04 .98- 1.11)	1.04 (0.97-1.11)	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.05 (0.98-1.12)	1.02 (0.96-1.09)	1.04 (0.98-1.10)	1.04 (0.97-1.11)	
1.12 .82-1.53) 0.98^{1} .79-1.20) 0.98^{2} .45-2.10)	1.12 (0.82-1.54) 0.98^{1} (0.79-1.20) 0.98^{2} (0.45-2.11)	1.10 (0.81-1.51) 1.05^{1} (0.86-1.30) 0.95^{2} (0.44-2.05)	1.11 (0.81-1.51) 1.05^{1} (0.86-1.30) 0.95^{2} (0.44-2.05)	1.09 (0.80-1.49) 1.05^{1} (0.85-1.29) 0.96^{2} (0.44-2.06)	1.09 (0.80-1.49) 1.05^{1} (0.85-1.29) 0.96^{2} (0.44-2.07)	1.17 (0.86-1.59) 0.95^{1} (0.77-1.16) 1.06^{2} (0.50-2.20)	1.20 (0.89-1.60) 0.96^{1} (0.77-1.17) 1.18^{2} (0.57-2.40)		
*1.15 .03-1.29)	*1.15 (1.03-1.29)	*1.16 (1.04-1.30)	*1.16 (1.04-1.30)	*1.18 (1.05-1.32)	*1.18 (1.05-1.32)	*1.18 (1.06-1.31)			
*4.56 .77-5.51)	*4.56 (3.77-5.51)	*4.58 (3.80-5.54)	*4.59 (3.80-5.55)	*4.62 (3.82-5.57)	*4.61 (3.82-5.57)	*4.69 (3.91-5.62)			
*1.67 .25-2.22)	*1.67 (1.25-2.22)	*1.66 (1.25-2.21)	*1.67 (1.25-2.25)	*1.66 (1.25-2.21)	*2.00 (1.76-2.26)	*1.43 (1.28-1.60)			
1.00 .69-1.45)	1.00 (0.70-1.45)	1.03 (0.72-1.48)	1.03 (0.72-1.48)	1.04 (0.72-1.49)	1.04 (0.72-1.50)	1.07 (0.75-1.52)			
1.05 .66-1.68)	1.05 (0.66-1.67)	1.03 (0.65-1.64)	1.02 (0.64-1.63)	1.03 (0.64-1.63)	1.03 (0.65-1.65)	1.12 (0.72-1.75)			
*1.75 .06-2.88)	*1.76 (1.07-2.89)	*1.79 (1.08-2.94)	*1.78 (1.08-2.93)	*1.78 (1.08-2.92)	*1.77 (1.08-2.92)	*1.92 (1.19-3.09)			
0.95 .80-1.11)	0.95 (0.80-1.11)	0.94 (0.80-1.11)	0.94 (0.80-1.11)	0.94 (0.80-1.10)	0.94 (0.80-1.10)				
*4.71 .09-5.42)	*4.71 (4.09-5.42)	*4.71 (4.09-5.42)	*4.71 (4.09-5.42)	*4.84 (4.21-5.57)	*4.83 (4.20-5.56)				
*4.24 .63-4.95)	*4.24 (3.63-4.95)	*4.24 (3.63-4.95)	*4.24 (3.63-4.95)	*4.25 (3.65-4.97)	*4.25 (3.65-4.97)				
1.04 .88-1.23)	1.04 (0.88-1.23)	1.04 (0.88-1.22)	1.04 (0.88-1.22)	1.14 (0.97-1.33)	1.14 (0.98-1.33)				
*1.40 .07-1.84)	*1.40 (1.07-1.84)	*1.38 (1.05-1.81)	*1.39 (1.06-1.82)	*1.40 (1.06-1.83)	*1.65 (1.43-1.90)				
1.21 .89-1.66)	1.21 (0.89-1.66)	1.24 (0.91-1.70)	1.24 (0.91-1.70)	1.24 (0.91-1.69)					
0.98 .86-1.12)	0.98 (0.86-1.12)	0.98 (0.86-1.12)	1.01 (0.89-1.15)						
1.01 .71-1.43)	1.01 (0.71-1.43)	1.02 (0.72-1.44)	1.01 (0.71-1.42)						
0.69 .41-1.14)	0.69 (0.41-1.14)	0.67 (0.44-1.11)	1.06 (0.83-1.36)						
1.25 .89-1.76)	1.25 (0.89-1.76)	1.25 (0.90-1.75)	1.32 (0.95-1.84)						
1.21 .81-1.76)	1.21 (0.81-1.80)	1.19 (0.80-1.78)	1.13 (0.76-1.68)						
*1.87 1.05-3.33)	*1.88 (1.06-3.34)	*1.89 (1.06-3.35)							
1.58 .85-2.91)	1.57 (0.85-2.90)								
*2.35 1.60-3.45)	*2.36 (1.61-3.45)								
*2.59 1.76-3.79)	*2.58 (1.76 -3.79)								
*6.57 .63-16.40)	*6.64 (2.66-16.50)								
1.27 .99-1.63)	1.27 (0.99-1.63)								
1.26 .59-2.66)									
1.10 .68-1.78)									

Appendix G: Adjusted Odds Ratios for the General Model of Maternal Risk Factors and Low Birth Weight

Risk Factor	17	16	15	14	13	12	11	10	9	8
Age Younger Age^1 Older Age^2	0.88^1 (0.68-1.16) 1.13^2 (0.91-1.41)	0.86^1 (0.69-1.09) 1.19^2 (0.98-1.44)	0.85^1 (0.68-1.08) 1.19^2 (0.99-1.44)	0.85^1 (0.68-1.07) *1.22^2 (1.01-1.47)	0.84^1 (0.67-1.05) *1.23^2 (1.02-1.48)	0.83^1 (0.66-1.05) *1.23^2 (1.10-1.56)	*0.77^1 (0.62-.0.97) *1.31^2 (1.10-1.56)	*0.77^1 (0.61-0.96) *1.31^2 (1.10-1.56)	*0.78^1 (0.62-0.97) *1.33^2 (1.12-1.58)	*0.79^1 (0.63-.0.98) *1.33^2 (1.12-1.58)
Genetic Ht <152cm Wt$\geq$91kg^1 Wt$\leq$45 kg^2	1.47 (0.99-2.19) *0.42^1 (0.30-0.59) 1.93^2 (0.88-4.23)	1.35 (0.95-1.90) *0.48^1 (0.36-0.65) 1.57^2 (0.76-3.24)	1.35 (0.95-1.91) *0.48^1 (0.35-0.64) 1.56^2 (0.75-3.22)	1.39 (0.99-1.96) *0.48^1 (0.36-0.64) 1.49^2 (0.72-3.06)	1.37 (0.97-1.93) *0.49^1 (0.36-0.65) 1.48^2 (0.72-3.02)	1.39 (0.99-1.96) *0.48^1 (0.36-0.64) 1.41^2 (0.77-2.87)	*1.42 (1.01-1.99) *0.49^1 (0.36-0.65) 1.45^2 (0.72-2.93)	1.37 (0.98-1.92) *0.56^1 (0.42-0.75) 1.62^2 (0.81-3.23)	1.38 (0.99-1.92) *0.59^1 (0.44-0.77) 1.62^2 (0.82-3.17)	1.37 (0.99-1.91) *0.58^1 (0.44-0.77) 1.78^2 (0.91-3.46)
Obstetrical History Abortion	1.03 (0.89-1.21)	1.07 (0.93-1.22)	1.07 (0.93-1.22)	1.08 (0.95-1.23)	1.09 (0.96-1.24)	1.10 (0.96-1.25)	1.10 (0.96-1.25)	1.10 (0.96-1.25)	1.18 (0.98-1.27)	1.12 (0.98-1.27)
Preterm Birth	*2.31 (1.77-3.01)	*4.13 (3.32-5.15)	*4.11 (3.30-5.12)	*4.21 (3.39-5.22)	*4.20 (3.39-5.20)	*4.26 (3.44-5.27)	*4.23 (3.42-5.23)	*4.25 (3.44-5.25)	*4.29 (3.49-5.28)	*4.27 (3.47-5.25)
Nulliparity	1.47 (0.99-2.78)	*1.70 (1.19-2.42)	*1.68 (1.18-2.40)	*1.63 (1.15-2.32)	*1.63 (1.15-2.32)	*1.66 (1.17-2.36)	*1.65 (1.16-2.34)	*1.75 (1.23-2.47)	*1.88 (1.33-2.65)	*1.87 (1.32-2.63)
Stillbirth	1.10 (0.66-1.83)	1.05 (0.70-1.57)	1.05 (0.70-1.58)	1.10 (0.74-1.64)	1.08 (0.73-1.61)	1.10 (0.74-1.63)	1.10 (0.74-1.63)	1.07 (0.72-1.59)	1.16 (0.79-1.69)	1.16 (0.79-1.70)
Neonatal Death	1.15 (0.61-2.15)	1.16 (0.69-1.94)	1.15 (0.68-1.93)	1.23 (0.74-2.04)	1.24 (0.75-2.04)	1.23 (0.74-2.03)	1.20 (0.72-1.99)	1.22 (0.74-2.02)	1.21 (0.74-1.99)	1.22 (0.74-2.00)
SGA Birth	*8.73 (5.49-13.90)	*5.88 (3.90-8.86)	*5.85 (3,88-8.81)	*5.58 (3.73-8.36)	*5.61 (3.74-8.40)	*5.57 (3.72-8.34)	*5.56 (3.71-8.33)	*5.55 (3.70-8.31)	*6.44 (4.33-9.59)	*6.70 (4.52-9.95)
Prenatal Care Missing Visits Data	0.87 (0.69-1.09)	0.91 (0.75-1.10)	0.91 (0.75-1.10)	0.89 (0.74-1.08)	0.89 (0.74-1.07)	0.88 (0.73-1.06)	0.88 (0.73-1.06)	0.88 (0.73-1.05)	0.87 (0.72-1.04)	0.87 (0.73-1.04)
≤ 4 Visits	*2.07 (1.71-2.51)	*4.00 (3.40-4.71)	*4.04 (3.43-4.74)	*4.22 (3.60-4.95)	*4.26 (3.63-5.00)	*4.30 (3.67-5.04)	*4.28 (3.65-5.02)	*4.28 (3.65-5.01)	*4.23 (3.62-4.93)	*4.20 (3.60-4.90)
5-7 Visits	*1.74 (1.40-2.17)	*3.18 (2.64-3.84)	*3.20 (2.65-3.86)	*3.30 (2.75-3.98)	*3.29 (2.73-3.96)	*3.34 (2.78-4.01)	*3.30 (2.75-3.97)	*3.29 (2.74-3.95)	*3.20 (2.67-3.83)	*3.18 (2.66-3.81)
Missing Classes Data	1.03 (0.83-1.28)	1.05 (0.87-1.27)	1.05 (0.87-1.27)	1.02 (0.85-1.24)	1.02 (0.85-1.23)	1.03 (0.85-1.24)	1.03 (0.86-1.24)	1.05 (0.87-1.26)	1.06 (0.88-1.27)	1.05 (0.88-1.26)
No Classes	1.09 (0.75-1.59)	1.33 (0.95-1.86)	1.33 (0.95-1.86)	1.32 (0.95-1.84)	1.32 (0.95-1.84)	1.33 (0.96-1.86)	1.32 (0.95-1.84)	1.30 (0.93-1.81)	1.34 (0.96-1.86)	1.34 (0.97-1.86)
Classes x Parity Interaction	*1.53 (1.00-2.33)	1.45 (0.99-2.12)	*1.46 (1.00-2.13)	*1.52 (1.04-2.21)	*1.51 (1.04-2.20)	*1.51 (1.04-2.20)	*1.52 (1.04-2.21)	*1.57 (1.05-2.21)	*1.44 (1.00-2.09)	*1.45 (1.00-2.10)
Lifestyle Smokers	*2.02 (1.73-2.39)	*1.63 (1.41-1.87)	*1.63 (1.42-1.88)	*1.63 (1.42-1.87)	*1.63 (1.42-1.87)	*1.62 (1.41-1.86)	*1.63 (1.42-1.87)	*1.57 (1.37-1.80)	*1.56 (1.37-1.79)	*1.57 (1.37-1.79)
Quitters	0.93 (0.59-1.45)	0.92 (0.62-1.36)	0.91 (0.61-1.36)	0.96 (0.65-1.42)	0.97 (0.66-1.42)	0.98 (0.67-1.44)	0.97 (0.66-1.42)	1.01 (0.69-1.47)	1.01 (0.70-1.47)	1.01 (0.70-1.47)
Alcohol Use	0.62 (0.29-1.35)	*0.44 (0.20-0.94)	*0.44 (0.21-0.95)	*0.45 (0.21-0.95)	*0.46 (0.22-0.97)	*0.47 (0.22-0.99)	0.48 (0.23-1.00)	*0.47 (0.23-0.98)	*0.47 (0.23-0.98)	*0.48 (0.23-.0.99)
Street Drug Use	1.41 (0.96-2.06)	*1.51 (1.10-2.08)	*1.51 (1.10-2.07)	*1.55 (1.14-2.12)	*1.53 (1.12-2.09)	*1.54 (1.12-2.10)	*1.54 (1.13-2.11)	*1.55 (1.14-2.12)	*1.58 (1.17-2.14)	*1.58 (1.17-2.14)
Missing Street Drug Use Data	0.87 (0.54-1.43)	0.96 (0.64-1.45)	0.98 (0.65-1.47)	0.96 (0.65-1.43)	0.97 (0.65-1.44)	0.97 (0.65-1.44)	0.97 (0.65-1.45)	0.96 (0.65-1.43)	0.98 (0.67-1.43)	0.98 (0.67-1.43)
Alcohol x Smoking Interaction	1.60 (0.69-3.69)	*2.69 (1.20-6.02)	*2.62 (1.17-5.87)	*2.56 (1.16-5.66)	*2.53 (1.15-5.57)	*2.45 (1.12-5.36)	*2.43 (1.15-5.33)	*2.43 (1.12-5.27)	*2.46 (1.14-5.30)	*2.50 (1.16-5.37)
Pre-existing Disease Heart Disease	1.36 (0.58-3.17)	1.53 (0.75-3.10)	1.51 (0.74-3.07)	1.53 (0.77-3.05)	1.55 (0.78-3.09)	1.51 (0.75-3.02)	1.50 (0.75-3.01)	1.50 (0.75-2.99)	1.41 (0.70-2.81)	1.38 (0.69-2.75)
Diabetes Mellitus	0.61 (0.32-1.19)	1.15 (0.66-1.97)	1.14 (0.66-1.98)	1.09 (0.63-1.88)	1.07 (0.62-1.85)	1.10 (0.64-1.88)	1.12 (0.66-1.92)	1.14 (0.67-1.95)	1.34 (0.81-2.23)	1.38 (0.83-2.29)
Chronic Hypertension	*2.11 (1.23-3.62)	*2.25 (1.45-3.45)	*2.24 (1.45-3.47)	*2.15 (1.39-3.32)	*2.19 (1.42-3.38)	*2.16 (1.41-3.28)	*2.20 (1.44-3.33)	*3.70 (2.49-5.51)	*3.95 (2.69-5.80)	*3.91 (2.66-5.75)
Renal Disease	2.03 (0.55-7.48)	*4.09 (1.39-12.05)	*4.07 (1.37-12.05)	*4.21 (1.47-12.05)	*4.09 (1.44-11.56)	*4.82 (1.80-12.90)	*5.02 (1.90-13.24)	*5.96 (2.35-15.10)	*6.31 (2.58-15.44)	*6.61 (2.71-16.14)
Other Medical Diseases	0.99 (0.71-1.40)	1.18 (0.88-1.60)	1.17 (0.87-1.56)	1.15 (0.86-1.53)	1.16 (0.87-1.54)	1.18 (0.89-1.57)	1.18 (0.89-1.57)	1.19 (0.90-1.58)	1.17 (0.89-1.55)	1.20 (0.91-1.59)
Nutrition Anemia	*2.40 (1.10-5.24)	1.94 (0.96-3.91)	1.96 (0.97-3.95)	1.90 (0.96-3.78)	*1.98 (1.00-3.92)	*1.97 (1.00-3.90)	1.92 (0.97-3.82)	1.92 (0.97-3.79)	1.88 (0.96-3.68)	
Poor Gestational Weight Gain	*2.43 (1.50-3.91)	*2.01 (1.33-3.03)	*2.00 (1.32-3.01)	*1.99 (1.32-2.99)	*1.98 (1.32-2.98)	*1.96 (1.31-2.94)	*1.95 (1.30-2.93)	*2.02 (1.35-3.02	*2.33 (1.58-3.46)	
Problems in Current Pregnancy Acute Medical Problems	1.42 (0.63-3.19)	*2.09 (1.04-4.20)	*2.12 (1.06-4.25)	*2.30 (1.17-4.50)	*2.27 (1.15-4.44)	*2.28 (1.16-4.47)	*2.28 (1.17-4.48)	*2.13 (1.09-4.18)		
Poly/Oligo hydramnios	*5.78 (4.40-7.59)	*7.46 (5.97-9.33)	*7.45 (5.97-9.30)	*7.62 (6.13-9.47)	*7.74 (6.23-9.60)	*7.65 (6.17-9.49)	*7.64 (6.16-9.48)	*7.50 (6.06-9.28)		
Gestational Diabetes	0.97 (0.66-1.42)	1.12 (0.81-1.56)	1.12 (0.81-1.55)	1.13 (0.82-1.56)	1.13 (0.82-1.56)	1.13 (0.82-1.55)	1.13 (0.82-1.55)	1.22 (0.89-1.67)		
Complications Gestational Hypertension	*2.05 (1.41-2.97)	*2.35 (1.71-3.21)	*2.34 (1.71-3.21)	*2.23 (1.63-3.05)	*2.19 (1.60-3.00)	*3.59 (2.81-4.60)	*3.67 (2.95-4.56)			
Gest Hyp x Age Interaction Younger^1 Older^2	*0.16^1 (0.03-0.68) 1.40^2 (0.69-2.80)	*0.17^1 (0.05-0.55) *1.77^2 (1.00-3.12)	*0.18^1 (0.05-0.56) *1.77^2 (1.00-3.12)	*0.16^1 (0.05-0.52) 1.73^2 (0.99-3.04)	*0.17^1 (0.05-0.54) 1.72^2 (0.98-3.01)	*0.27^1 (0.09-0.80) *1.79^2 (1.05-3.06)				
Pre-eclampsia	*2.20 (1.32-3.68)	*3.76 (2.48-5.70)	*3.77 (2.49-5.71)	*3.79 (2.51-5.74)	*3.77 (2.49-5.70)					
Bleeding < 20 weeks	*1.49 (1.11-2.01)	*1.80 (1.40-2.32)	*1.85 (1.44-2.37)	*2.29 (1.80-2.92)						
Bleeding ≥ 20 weeks	*2.12 (1.62-2.79)	*4.65 (3.73-5.80)	*5.06 (4.09-6.26)							
Placenta Previa	0.90 (0.47-1.73)	*2.73 (1.53-4.89)								
Gestational Age	*31.28 (26.91-36.36)									

*Significant $p < 0.05$

2

	10	9	8	7	6	5	4	3	2	1
	*0.77^1 (0.61-0.96) *1.31^2 (1.10-1.56)	*0.78^1 (0.62-0.97) *1.33^2 (1.12-1.58)	*0.79^1 (0.63- 0.98) *1.33^2 (1.12-1.58)	*0.77 (0.62-.0.95) *1.35 (1.14-1.61)	*0.76^1 (0.61-.0.95) *1.35^2 (1.14-1.60)	0.94^1 (0.76-1.17) *1.26^2 (1.06-.1.50)	0.96^1 (0.78-1.18) *1.26^2 (1.07-1.50)	*1.41^1 (1.15-1.73) *1.18^2 (1.00-1.39)	*1.56^1 (1.28-1.91) 1.15^2 (0.98-1.35)	*1.57^1 (1.29-1.91) 1.16^2 (0.99-1.37)
	1.37 (0.98-1.92) *0.56^1 (0.42-0.75) 1.62^2 (0.81-3.23)	1.38 (0.99-1.92) *0.59^1 (0.44-0.77) 1.62^2 (0.82-3.17)	1.37 (0.99-1.91) *0.58^1 (0.44-0.77) 1.78^2 (0.91-3.46)	1.36 (0.98-1.89) *0.65 (0.50-0.86) 1.74^2 (0.89 -3.38)	1.37 (0.99-1.90) *0.65^1 (0.50-0.86) 1.75^2 (0.90 -3.40)	1.24 (0.89-1.72) *0.65^1 (0.50-0.86) 1.78^2 (0.91-3.46)	1.25 (0.90-1.73) *0.65 (0.50-0.86) 1.78^2 (0.91-3.46)	*1.39 (1.02-1.92) *0.61^1 (0.46-0.79) *1.90^2 (1.00-3.63)	*1.44 (1.05-1.97) *0.63^1 (0.48-0.81) *2.20^2 (1.20-4.05)	
	1.10 (0.96-1.25)	1.18 (0.98-1.27)	1.12 (0.98-1.27)	1.12 (0.98-1.27)	1.12 (0.98-1.27)	*1.21 (1.07-1.37)	*1.21 (1.07-1.37)	*1.23 (1.09-1.40)		
	*4.25 (3.44-5.25)	*4.29 (3.49-5.28)	*4.27 (3.47-5.25)	*4.33 (3.52-5.32)	*4.34 (3.53-5.34)	*4.57 (3.72-5.61)	*4.56 (3.72-5.60)	*4.88 (4.00-5.96)		
	*1.75 (1.23-2.47)	*1.88 (1.33-2.65)	*1.87 (1.32-2.63)	*1.86 (1.32-2.62)	*1.87 (1.32-2.63)	*1.86 (1.32-2.62)	*2.59 (2.25-2.97)	*1.76 (1.56-2.00)		
	1.07 (0.72-1.59)	1.16 (0.79-1.69)	1.16 (0.79-1.70)	1.19 (0.81-1.74)	1.19 (0.81-1.74)	1.23 (0.84-1.80)	1.24 (0.85-1.81)	1.28 (0.88-1.85)		
	1.22 (0.74-2.02)	1.21 (0.74-1.99)	1.22 (0.74-2.00)	1.19 (0.73-1.95)	1.18 (0.72-1.94)	1.18 (0.72-1.93)	1.19 (0.73-1.96)	1.29 (0.80-2.08)		
	*5.55 (3.70-8.31)	*6.44 (4.33-9.59)	*6.70 (4.52-9.95)	*6.79 (4.57-10.06)	*6.80 (4.58-10.08)	*7.08 (4.79-10.46)	*7.05 (4.77-10.42)	*7.17 (4.95-10.39)		
	0.88 (0.73-1.05)	0.87 (0.72-1.04)	0.87 (0.73-1.04)	0.86 (0.72-1.03)	0.86 (0.72-1.03)	0.83 (0.69-1.00)	0.83 (0.69-1.00)			
	*4.28 (3.65-5.01)	*4.23 (3.62-4.93)	*4.20 (3.60-4.90)	*4.21 (3.61-4.91)	*4.21 (3.61-4.91)	*4.61 (3.96-5.36)	*4.60 (3.95-5.35)			
	*3.29 (2.74-3.95)	*3.20 (2.67-3.83)	*3.18 (2.66-3.81)	*3.19 (2.67-3.82)	*3.19 (2.67-3.83)	*3.33 (2.78-3.98)	*3.33 (2.78-3.97)			
	1.05 (0.87-1.26)	1.06 (0.88-1.27)	1.05 (0.88-1.26)	1.05 (0.88-1.26)	1.05 (0.88-1.26)	1.14 (0.97-1.35)	1.15 (0.97-1.36)			
	1.30 (0.93-1.81)	1.34 (0.96-1.86)	1.34 (0.97-1.86)	1.31 (0.95-1.82)	1.33 (0.96-1.84)	*1.44 (1.04-1.99)	*1.95 (1.66-2.29)			
	*1.57 (1.05-2.21)	*1.44 (1.00-2.09)	*1.45 (1.00-2.10)	*1.50 (1.04-2.17)	*1.50 (1.04-2.16)	*1.46 (1.01-2.10)				
	*1.57 (1.37-1.80)	*1.56 (1.37-1.79)	*1.57 (1.37-1.79)	*1.56 (1.36-1.78)	*1.61 (1.41-1.83)					
	1.01 (0.69-1.47)	1.01 (0.70-1.47)	1.01 (0.70-1.47)	1.02 (0.70-1.48)	1.00 (0.70-1.47)					
	*0.47 (0.23-0.98)	*0.47 (0.23-0.98)	*0.48 (0.23-.0.99)	0.49 (0.24-1.01)	1.03 (0.80-1.32)					
	*1.55 (1.14-2.12)	*1.58 (1.17-2.14)	*1.58 (1.17-2.14)	*1.58 (1.17-2.13)	*1.66 (1.23-2.23)					
	0.96 (0.65-1.43)	0.98 (0.67-1.43)	0.98 (0.67-1.43)	0.96 (0.66-1.41)	0.92 (0.63-1.34)					
	*2.43 (1.12-5.27)	*2.46 (1.14-5.30)	*2.50 (1.16-5.37)	*2.42 (1.13-5.17)						
	1.50 (0.75-2.99)	1.41 (0.70-2.81)	1.38 (0.69-2.75)							
	1.14 (0.67-1.95)	1.34 (0.81-2.23)	1.38 (0.83-2.29)							
	*3.70 (2.49-5.51)	*3.95 (2.69-5.80)	*3.91 (2.66-5.75)							
	*5.96 (2.35-15.10)	*6.31 (2.58-15.44)	*6.61 (2.71-16.14)							
	1.19 (0.90-1.58)	1.17 (0.89-1.55)	1.20 (0.91-1.59)							
	1.92 (0.97-3.79)	1.88 (0.96-3.68)								
	*2.02 (1.35-3.02)	*2.33 (1.58-3.46)								
	*2.13 (1.09-4.18)									
	*7.50 (6.06-9.28)									
	1.22 (0.89-1.67)									

Appendix H: Adjusted Odds Ratios for the Model of Maternal Risk Factors, Socio-economic Status, and Low Birth Weight

Risk Factor	**18**	**17**	**16**	**15**	**14**	**13**	**12**	**11**	**10**	**9**	
Age Younger Age^{1} Older Age^{2}	0.79^{1} (0.58-1.05) 1.12^{2} (0.89-1.40)	0.80^{1} (0.68-1.00) 1.16^{2} (0.95-1.40)	0.79^{1} (0.61-1.02) 1.16^{2} (0.95-1.41)	0.79^{1} (0.61-1.02) 1.18^{2} (0.97-1.44)	*0.77^{1} (0.60-0.99) 1.20^{2} (0.98-1.45)	*0.77^{1} (0.60-0.99) 1.20^{2} (0.98-1.45)	*0.73^{1} (0.57-0.93) *1.28^{2} (1.07-1.54)	*0.72^{1} (0.56-0.96) *1.28^{2} (1.07-1.54)	*0.73^{1} (0.57-0.92) *1.31^{2} (1.10-1.57)	*0.74^{1} (0.58-0.94) *1.31^{2} (1.10-1.56)	(0. (1.
SES Fac1_1	1.06 (0.98-1.14)	1.01 (0.95-1.10)	1.01 (0.94-1.08)	1.01 (0.94-1.07)	1.00 (0.94-1.07)	1.00 (0.94-1.07)	1.00 (0.94-1.07)	1.00 (0.93-1.06)	0.99 (0.93-1.06)	0.99 (0.93-1.06)	(0.
Fac2_2	1.02 (0.92-1.12)	1.04 (0.95-1.10)	1.03 (0.95-1.12)	1.03 (0.95-1.12)	1.03 (0.95-1.12)	1.04 (0.95-1.12)	1.03 (0.95-1.12)	1.02 (0.94-1.11)	1.02 (0.94-1.10)	1.02 (0.94-1.10)	(0.
Genetic Ht < 152 cm Wt ≥ 91kg^{1} Wt ≤ 45kg^{2}	1.40 (0.92-2.12) *0.38^{1} (0.26-0.55) *2.51^{2} (1.15-5.44)	1.33 (0.93-1.90) *0.44^{1} (0.32-0.61) 1.98^{2} (0.97-4.10)	1.33 (0.93-1.91) *0.44^{1} (0.32-0.60) 1.97^{2} (0.96-4.04)	1.38 (0.97-1.97) *0.44^{1} (0.32-0.60) 1.88^{2} (0.92-3.84)	1.36 (0.95-1.94) *0.45^{1} (0.33-0.61) 1.82^{2} (0.89-3.72)	1.38 (0.97-1.97) *0.45^{1} (0.33-0.61) 1.76^{2} (0.87-3.56)	1.40 (0.99-1.99) *0.45^{1} (0.33-0.61) 1.77^{2} (0.87-3.57)	1.35 (0.95-1.92) *0.53^{1} (0.39-0.72) 1.94^{2} (0.97-3.87)	1.36 (0.96-1.92) *0.55^{1} (0.41-0.71) 1.91^{2} (0.97-3.73)	1.36 (0.97-1.91) *0.55^{1} (0.41-0.74) *2.06^{2} (1.05-4.02)	(0. (0. (1.
Obstetrical History Abortion	1.01 (0.86-1.18)	1.05 (0.91-1.20)	1.05 (0.91-1.20)	1.06 (0.92-1.22)	1.07 (0.93-1.23)	1.08 (0.94-1.24)	1.08 (0.94-1.24)	1.08 (0.94-1.23)	1.10 (0.96-1.26)	1.10 (0.97-1.26)	(0.
Preterm Birth	*2.43 (1.85-3.21)	*4.41 (3.50-5.50)	*4.38 (3.49-5.51)	*4.47 (3.58-5.60)	*4.47 (3.58-5.59)	*4.52 (3.62-5.65)	*4.49 (3.60-5.61)	*4.52 (3.62-5.64)	*4.46 (3.59-5.54)	*4.44 (3.38-5.51)	(3.
Nulliparity	1.40 (0.94-2.09)	*1.60 (1.12-2.30)	*1.58 (1.11-2.26)	*1.53 (1.08-2.18)	*1.53 (1.07-2.18)	*1.56 (1.10-2.23)	*1.55 (1.09-2.20)	*1.65 (1.16-2.34)	*1.77 (1.25-2.51)	*1.76 (1.25-2.50)	(1.
Stillbirth	1.10 (0.66-1.86)	1.09 (0.72-1.67)	1.10 (0.72-1.67)	1.17 (0.77-1.76)	1.14 (0.75-1.72)	1.16 (0.77-1.74)	1.15 (0.77-1.73)	1.12 (0.75-1.69)	1.20 (0.81-1.78)	1.22 (0.82-1.80)	(0
Neonatal Death	1.30 (0.66-2.47)	1.16 (0.68-1.97)	1.16 (0.68-1.97)	1.22 (0.73-2.06)	1.23 (0.73-2.07)	1.23 (0.73-2.07)	1.20 (0.71-2.02)	1.23 (0.73-2.06)	1.26 (0.75-2.09)	1.25 (0.75-2.08)	(0.
SGA Birth	*8.63 (5.32-14.00)	*6.12 (4.01-9.30)	*6.08 (3.99-9.27)	*5.81 (3.84-8.80)	*5.81 (3.83-8.81)	*5.76 (3.80-8.73)	*5.76 (3.79-8.73)	*5.78 (3.81-8.76)	*6.73 (4.47-10.12)	*6.99 (4.46-10.49)	(4.
Prenatal Care Missing Visits Data	0.86 (0.67-1.10)	0.92 (0.75-1.10)	0.91 (0.74-1.11)	0.90 (0.74-1.10)	0.89 (0.73-1.09)	0.88 (0.72-1.08)	0.88 (0.72-1.10)	0.88 (0.72-1.07)	0.86 (0.71-1.05)	0.87 (0.72-1.05)	(0
≤4 Visits	*2.04 (1.66-2.50)	*3.90 (3.28-4.60)	*3.94 (3.32-4.67)	*4.12 (3.48-4.87)	*4.17 (3.52-4.93)	*4.20 (3.55-4.97)	*4.18 (3.54-4.94)	*4.20 (3.55-4.96)	*4.17 (3.55-4.90)	*4.14 (3.52-4.87)	(3
5-7 Visits	*1.86 (1.49-2.33)	*3.45 (2.85-4.20)	*3.48 (2.87-4.21)	*3.57 (2.95-4.32)	*3.54 (2.93-4.28)	*3.58 (2.96-4.33)	*3.56 (2.95-4.30)	*3.53 (2.93-4.26)	*3.42 (2.84-4.11)	*3.40 (2.83-4.10)	(2.
Missing Classes Data	1.03 (0.82-1.30)	1.02 (0.84-1.20)	1.02 (0.84-1.25)	1.00 (0.82-1.22)	1.00 (0.82-1.22)	1.00 (0.82-1.22)	1.00 (0.82-1.22)	1.01 (0.83-1.23)	1.02 (0.84-1.24)	1.01 (0.84-1.23)	(0.
No Classes	1.02 (0.70-1.49)	1.22 (0.87-1.71)	1.22 (0.87-1.71)	1.21 (0.87-1.70)	1.21 (0.86-1.69)	1.23 (0.88-1.72)	1.23 (0.87-1.70)	1.20 (0.86-1.68)	1.23 (0.89-1.72)	1.24 (0.89-1.70)	(0
Classes x Parity Interaction	*1.58 (1.02-2.43)	*1.56 (1.06-2.30)	*1.57 (1.07-2.31)	*1.64 (1.12-2.41)	*1.64 (1.12-2.40)	*1.63 (1.12-2.39)	*1.64 (1.12-2.40)	*1.65 (1.13-2.41)	*1.56 (1.07-2.27)	*1.57 (1.08-2.28)	(1.
Lifestyle Smokers	*2.07 (1.75-2.45)	*1.65 (1.43-1.91)	*1.66 (1.43-1.92)	*1.66 (1.43-1.92)	*1.65 (1.43-1.91)	*1.64 (1.42-1.90)	*1.66 (1.43-1.91)	*1.60 (1.38-1.84)	*1.60 (1.39-1.84)	*1.60 (1.39-1.84)	(1
Quitters	0.98 (0.61-1.56)	0.96 (0.63-1.42)	0.95 (0.63-1.44)	1.01 (0.68-1.51)	1.02 (0.69-1.52)	1.04 (0.70-1.55)	1.04 (0.70-1.55)	1.06 (0.71-1.57)	1.03 (0.70-1.52)	1.04 (0.70-1.52)	(0
Alcohol Use	0.64 (0.29-1.39)	0.49 (0.23-1.00)	0.49 (0.23-1.05)	0.50 (0.24-1.06)	0.52 (0.25-1.08)	0.51 (0.24-1.08)	0.52 (0.25-1.08)	0.51 (0.24-1.05)	0.52 (0.25-1.06)	0.52 (0.25-1.07)	(0
Use Street Drugs	*1.71 (1.13-2.59)	*1.65 (1.16-2.33)	*1.65 (1.16-2.33)	*1.70 (1.21-2.40)	*1.69 (1.20-2.39)	*1.71 (1.21-2.41)	*1.73 (1.23-2.44)	*1.72 (1.22-2.41)	*1.69 (1.21-2.36)	*1.69 (1.29-2.36)	(1
Missing Street Drug Use Data	0.78 (0.47-1.32)	0.95 (0.61-1.40)	0.96 (0.62-1.49)	0.94 (0.61-1.44)	0.93 (0.61-1.43)	0.93 (0.60-1.42)	0.92 (0.60-1.41)	0.93 (0.60-1.42)	0.97 (0.64-1.47)	0.96 (0.64-1.46)	(0
Alcohol x Smoking Interaction	1.42 (0.60-3.35)	*2.25 (1.00-5.00)	2.20 (0.97-4.94)	2.18 (0.98-4.83)	2.15 (0.97-4.73)	2.13 (0.96-4.71)	2.12 (0.96-4.68)	2.15 (0.98-4.74)	2.09 (0.96-4.55)	2.12 (0.97-4.60)	(0
Pre-existing Disease Heart Disease	1.57 (0.66-3.76)	1.74 (0.85-3.60)	1.72 (0.84-3.54)	1.75 (0.87-3.51)	1.77 (0.88-3.57)	1.72 (0.85-3.49)	1.71 (0.85-3.47)	1.71 (0.86-3.47)	1.63 (0.81-3.30)	1.59 (0.79-3.19)	
Diabetes Mellitus	0.61 (0.31-1.20)	1.21 (0.69-2.10)	1.20 (0.69-2.09)	1.15 (0.66-1.99)	1.13 (0.65-1.96)	1.14 (0.64-1.96)	1.17 (0.68-2.00)	1.18 (0.61-2.02)	1.39 (0.83-2.30)	1.44 (0.87-2.40)	
Chronic Hypertension	*2.28 (1.31-3.97)	*2.34 (1.49-3.70)	*2.33 (1.49-3.65)	*2.23 (1.42-3.51)	*2.30 (1.47-3.59)	*2.26 (1.46-3.50)	*2.29 (1.48-3.54)	*3.84 (2.55-5.80)	*4.16 (2.80-6.20)	*4.12 (2.77-6.10)	
Renal Disease	1.84 (0.46-7.30)	*4.01 (1.21-13.00)	*3.99 (1.20-13.24)	*4.21 (1.32-13.40)	*4.09 (1.30-12.80)	*4.66 (1.57-13.80)	*4.87 (1.67-14.11)	*5.78 (2.08-16.00)	*6.40 (2.40-17.00)	*6.62 (2.50-17.40)	
Other Medical Diseases	0.97 (.678-1.38)	1.17 (0.86-1.60)	1.16 (0.85-1.57)	1.13 (0.84-1.54)	1.14 (0.84-1.55)	1.16 (0.86-1.57)	1.16 (0.86-1.56)	1.16 (0.86-1.57)	1.14 (0.80-1.50)	1.17 (0.87-1.60)	
Nutrition Anemia	*2.84 (1.23-6.51)	*2.22 (1.05-4.70)	*2.25 (1.06-4.75)	*2.13 (1.03-4.42)	*2.23 (1.08-4.59)	*2.23 (1.08-4.60)	*2.18 (1.06-4.52)	*2.21 (1.07-4.54)	*2.21 (1.09-4.47)		
Poor Gestational Weight Gain	*2.61 (1.59-4.27)	*2.08 (1.35-3.20)	*2.06 (1.34-3.17)	*2.06 (1.34-3.16)	*2.04 (1.32-3.13)	*2.02 (1.31-3.09)	*2.00 (1.30-3.08)	*2.04 (1.33-3.12)	*2.37 (156-3.59)		
Problems in Current Pregnancy Acute Medical Problems	1.48 (0.66-3.33)	*2.31 (1.15-4.70)	*2.34 (1.16-4.72)	*2.55 (1.29-5.02)	*2.52 (1.27-4.97)	*2.53 (1.28-5.00)	*2.54 (1.29-5.01)	*2.36 (1.20-4.67)			
Poly/Oligo hydramnios	*6.38 (4.82-8.45)	*8.06 (6.40-10.10)	*8.03 (6.38-10.10)	*8.20 (6.55-10.30)	*8.32 (6.60-10.40)	*8.24 (6.60-10.31)	*8.21 (6.57-10.20)	*8.03 (6.43-10.01)			
Gestational Diabetes	0.96 (0.65-1.41)	1.15 (0.82-1.60)	1.14 (0.81-1.60)	1.15 (0.83-1.60)	1.15 (0.83-1.60)	1.15 (0.83-1.59)	1.15 (0.83-1.59)	1.26 (0.92-1.73)			
Complications Gestational Hypertension	*2.21 (1.51-3.22)	*2.41 (1.74-3.30)	*2.41 (1.74-3.30)	*2.26 (1.63-3.13)	*2.22 (1.60-3.07)	*3.55 (2.75-4.58)	*3.76 (3.00-4.71)				
Gest Hyp x Age Interaction Younger^{1} Older^{2}	*0.18^{1} (0.04-0.86) 1.40^{2} (0.68-2.89)	*0.28^{1} (0.08-0.90) *1.88^{2} (1.04-3.40)	*0.28^{1} (0.08-.913) *1.88^{2} (1.04-3.40)	*0.26^{1} (0.08-0.87) *1.85^{2} (1.03-3.32)	*0.28^{1} (0.08-0.90) *1.84^{2} (1.03-3.28)	0.38^{1} (0.12-1.19) *1.89^{2} (1.08-3.29)					
Pre-eclampsia	*2.00 (1.18-3.40)	*3.49 (2.27-5.40)	*3.50 (2.27-5.40)	*3.56 (2.32-5.47)	*3.54 (2.31-5.43)						
Bleeding < 20 weeks	*1.55 (1.14-2.11)	*1.89 (1.46-2.40)	*1.94 (1.50-2.51)	*2.42 (1.89-3.10)							
Bleeding ≥ 20 weeks	*2.17 (1.64-2.88)	*4.70 (3.74-5.90)	*5.16 (4.14-6.44)								
Placenta Previa	0.91 (0.47-1.75)	*2.78 (1.54-5.00)									
Gestational Age	*31.90 (27.20-37.37)										

*Significant p < 0.05

11	10	9	8	7	6	5	4	3	2	1
*0.72^1 (0.56-0.96) *1.28^2 (1.07-1.54)	*0.73^1 (0.57-0.92) *1.31^2 (1.10-1.57)	*0.74^1 (0.58-0.94) *1.31^2 (1.10-1.56)	*0.72 (0.57-0.91) *1.33 (1.12-1.59)	*0.72 (0.56-0.91) *1.33 (1.12-1.59)	0.87^1 (0.69-1.10) *1.26^2 (1.06-1.51)	0.89^1 (0.71-1.12) *1.26^2 (1.06-1.51)	1.23^1 (0.98-1.54) *1.21^2 (1.02-1.44)	*1.30^1 (1.00-1.60) *1.18^2 (1.00-1.40)	*1.36^1 (1.09-1.70) *1.19^2 (1.01-1.41)	*1.45^1 (1.16-1.80) 1.17^2 (0.99-1.40)
1.00 (0.93-1.06)	0.99 (0.93-1.06)	0.99 (0.93-1.06)	1.00 (0.94-1.06)	1.00 (0.94-1.06)	1.04 (0.98-1.10)	1.04 (0.98-1.10)	*1.15 (1.08-1.22)	*1.17 (1.11-1.24)	*1.18 (1.11-1.25)	
1.02 (0.94-1.11)	1.02 (0.94-1.10)	1.02 (0.94-1.10)	1.02 (0.95-1.11)	1.02 (0.95-1.11)	1.02 (0.94-1.10)	1.02 (0.94-1.10)	1.00 (0.93-1.08)	1.03 (0.95-1.11)	1.03 (0.96-1.12)	
1.35 (0.95-1.92) *0.53^1 (0.39-0.72) 1.94^2 (0.97-3.87)	1.36 (0.96-1.92) *0.55^1 (0.41-0.71) 1.91^2 (0.97-3.73)	1.36 (0.97-1.91) *0.55^1 (0.41-0.74) *2.06^2 (1.05-4.02)	1.35 (0.96-1.89) *0.62 (0.46-0.83) *1.99^2 (1.02-3.89)	1.35 (0.96-1.90) *0.62 (0.46-0.82) *1.99^2 (1.02-3.89)	1.20 (0.86-1.69) *0.61^1 (0.46-0.82) *2.03^2 (1.05-4.00)	1.21 (0.86-1.70) *0.61^1 (0.46-0.82) *2.06^2 (1.05-4.01)	1.29 (0.93-1.08) *0.56 (0.42-0.75) *2.26^2 (1.18-4.34)	1.33 (0.96-1.84) *0.58^1 (0.44-0.78) *2.54^2 (1.37-4.70)		
1.08 (0.94-1.23)	1.10 (0.96-1.26)	1.10 (0.97-1.26)	1.10 (0.96-1.26)	1.10 (0.98-1.26)	*1.19 (1.04-1.36)	*1.19 (1.04-1.36)	*1.20 (1.05-1.36)			
*4.52 (3.62-5.64)	*4.46 (3.59-5.54)	*4.44 (3.38-5.51)	*4.50 (3.63-5.58)	*4.50 (3.63-5.59)	*4.68 (3.78-5.80)	*4.67 (3.77-5.78)	*4.89 (3.96-6.03)			
*1.65 (1.16-2.34)	*1.77 (1.25-2.51)	*1.76 (1.25-2.50)	*1.76 (1.24-2.49)	*1.76 (1.25-2.49)	*1.75 (1.24-2.47)	*2.59 (2.24-3.00)	*1.80 (1.58-2.05)			
1.12 (0.75-1.69)	1.20 (0.81-1.78)	1.22 (0.82-1.80)	1.25 (0.85-1.85)	1.26 (0.85-1.85)	1.30 (0.88-1.92)	1.31 (0.89-1.93)	1.34 (0.92-1.95)			
1.23 (0.73-2.06)	1.26 (0.75-2.09)	1.25 (0.75-2.08)	1.22 (0.73-2.03)	1.21 (0.73-2.01)	1.22 (0.73-2.02)	1.24 (0.74-2.06)	1.32 (0.81-2.16)			
*5.78 (3.81-8.76)	*6.73 (4.47-10.12)	*6.99 (4.46-10.49)	*7.07 (4.72-10.60)	*7.07 (4.72-10.60)	*7.37 (4.94-11.02)	*7.34 (4.91-10.97)	*7.30 (4.98-10.70)			
0.88 (0.72-1.07)	0.86 (0.71-1.05)	0.87 (0.72-1.05)	0.87 (0.71-1.05)	0.86 (0.71-1.05)	0.84 (0.69-1.01)	0.84 (0.66-1.02)				
*4.20 (3.55-4.96)	*4.17 (3.55-4.90)	*4.14 (3.52-4.87)	*4.13 (3.51-4.86)	*4.13 (3.51-4.86)	*4.42 (3.76-5.18)	*4.40 (3.75-5.17)				
*3.53 (2.93-4.26)	*3.42 (2.84-4.11)	*3.40 (2.83-4.10)	*3.41 (2.83-4.10)	*3.41 (2.84-4.10)	*3.52 (2.93-4.23)	*3.52 (2.93-4.22)				
1.01 (0.83-1.23)	1.02 (0.84-1.24)	1.01 (0.84-1.23)	1.01 (0.84-1.23)	1.01 (0.84-1.22)	1.13 (0.95-1.35)	1.15 (0.96-1.37)				
1.20 (0.86-1.68)	1.23 (0.89-1.72)	1.24 (0.89-1.70)	1.22 (0.88-1.69)	1.23 (0.88-1.70)	1.30 (0.94-1.81)	*1.87 (1.58-2.21)				
*1.65 (1.13-2.41)	*1.56 (1.07-2.27)	*1.57 (1.08-2.28)	*1.62 (1.12-2.35)	*1.62 (1.11-2.35)	*1.57 (1.08-2.28)					
*1.60 (1.38-1.84)	*1.60 (1.39-1.84)	*1.60 (1.39-1.84)	*1.59 (1.38-1.83)	*1.63 (1.42-1.87)						
1.06 (0.71-1.57)	1.03 (0.70-1.52)	1.04 (0.70-1.52)	1.04 (0.71-1.54)	1.03 (0.70-1.52)						
0.51 (0.24-1.05)	0.52 (0.25-1.06)	0.52 (0.25-1.07)	0.52 (0.25-1.07)	0.95 (0.73-1.26)						
*1.72 (1.22-2.41)	*1.69 (1.21-2.36)	*1.69 (1.29-2.36)	*1.67 (1.20-2.34)	*1.74 (1.25-2.42)						
0.93 (0.60-1.42)	0.97 (0.64-1.47)	0.96 (0.64-1.46)	0.96 (0.63-1.45)	0.92 (0.61-1.39)						
2.15 (0.98-4.74)	2.09 (0.96-4.55)	2.12 (0.97-4.60)	2.11 (0.97-4.57)							
1.71 (0.86-3.47)	1.63 (0.81-3.30)	1.59 (0.79-3.19)								
1.18 (0.61-2.02)	1.39 (0.83-2.30)	1.44 (0.87-2.40)								
*3.84 (2.55-5.80)	*4.16 (2.80-6.20)	*4.12 (2.77-6.10)								
*5.78 (2.08-16.00)	*6.40 (2.40-17.00)	*6.62 (2.50-17.40)								
1.16 (0.86-1.57)	1.14 (0.80-1.50)	1.17 (0.87-1.60)								
*2.21 (1.07-4.54)	*2.21 (1.09-4.47)									
*2.04 (1.33-3.12)	*2.37 (156-3.59)									
*2.36 (1.20-4.67)										
*8.03 (6.43-10.01)										
1.26 (0.92-1.73)										

Appendix I: Prevalence of Maternal Risk Factors Within Each Public Health Service Area

Risk Factor									Public Health S	
	Central % (N) (1809)	North Central % (N) (1866)	West Central % (N) (1883)	North East % (N) (2424)	West (North) % (N) (2147)	Millwoods % (N) (3506)	St Albert % (N) (1412)	Castle Downs % (N) (1306)	West (South) % (N) (1195)	So
Maternal Age										
12-19	11.3 (205)	6.5 (121)	7.3 (138)	8.7 (211)	8.2 (175)	5.0 (177)	3.4 (48)	5.1 (67)	5.4 (65)	
20-34	76.2 (1379)	82.3 (1536)	79.8 (1502)	81.8 (1982)	79.4 (1705)	83.8 (2935)	80.9 (1143)	83.2 (1086)	78.2 (934)	
35+	12.5 (225)	11.2 (209)	12.9 (243)	9.5 (231)	12.4 (267)	11.2 (394)	15.7 (221)	11.7 (153)	16.4 (196)	
Genetic and Constitutional										
Height<152 cm	5.4 (98)	2.9 (54)	3.8 (71)	2.6 (64)	1.6 (34)	2.2 (78)	1.6 (22)	3.2 (42)	1.5 (18)	
Weight ≤45 kg	0.2 (4)	0.1 (1)	0.3 (5)	0.3 (7)	0.6 (13)	0.8 (28)	0.1 (2)	0.2 (2)	0.3 (4)	
Weight ≥91 kg	6.7 (121)	7.7 (143)	6.6 (124)	9.9 (241)	6.3 (136)	7.9 (278)	4.4 (62)	6.9 (90)	5.4 (65)	
Obstetrical History										
Abortions 1or more	32.8 (593)	31.3 (584)	33.2 (626)	31.8 (772)	29.0 (623)	28.1 (985)	28.7 (405)	28.8 (376)	31.1 (372)	
Preterm Birth 1or more	5.7 (104)	4.9 (91)	3.7 (69)	4.5 (108)	3.4 (74)	3.7 (128)	4.3 (61)	4.1 (53)	3.1 (37)	
Stillbirth 1or more	2.0 (36)	1.9 (36)	1.1 (20)	1.5 (37)	1.2 (26)	1.0 (36)	1.3 (19)	1.4 (18)	1.5 (18)	
Neonatal Deaths	1.2 (22)	1.0 (19)	0.8 (16)	0.5 (12)	0.5 (10)	0.8 (29)	0.6 (8)	0.6 (8)	0.9 (11)	
SGA Birth	1.0 (18)	0.6 (11)	0.7 (14)	1.0 (24)	0.7 (15)	0.5 (18)	0.5 (7)	0.9 (12)	0.4 (5)	
Parity										
0	46.2 (836)	39.9 (744)	50.6 (952)	41.4 (1003)	43.0 (923)	41.1 (1440)	38.0 (537)	41.6 (544)	44.5 (532)	
1-3	49.2 (890)	56.9 (1061)	46.9 (884)	56.0 (1357)	54.9 (1173)	57.1 (2004)	60.6 (855)	56.0 (731)	53.7 (642)	
4+	4.6 (83)	3.2 (61)	2.5 (47)	2.6 (64)	2.1 (45)	1.8 (62)	1.4 (20)	2.4 (31)	1.8 (21)	
Prenatal Care										
Prenatal Classes										
Non Attendance	70.2 (1270)	67.2 (1254)	61.1 (1151)	63.8 (1548)	57.9 (1243)	51.3 (1800)	49.4 (697)	62.7 (819)	51.6 (617)	
Missing Classes Data	7.8 (142)	8.0 (150)	8.5 (160)	9.8 (237)	11.9 (256)	24.0 (841)	5.4 (76)	7.0 (92)	11.0 (131)	
Prenatal Visits										
≤4 visits	18.1 (327)	13.9 (259)	13.6 (256)	15.1 (365)	9.2 (198)	7.0 (244)	13.7 (194)	11.9 (155)	7.4 (89)	
Missing Visits Data	12.5 (226)	13.1 (244)	11.5 (216)	11.0 (267)	8.7 (187)	5.1 (179)	28.3 (400)	17.6 (230)	7.5 (90)	
5-7 visits	10.8 (196)	8.7 (163)	9.9 (186)	9.5 (256)	11.1 (239)	7.4 (260)	4.7 (67)	8.1 (106)	8.8 (105)	
Lifestyle Factors										
Smokers	37.4 (677)	29.8 (556)	28.0 (528)	35.4 (858)	32.4 (696)	23.0 (808)	18.1 (256)	23.4 (306)	20.5 (245)	
Alcohol Consumption	7.0 (126)	3.9 (72)	5.1 (96)	4.7 (114)	4.5 (97)	3.5 (121)	6.0 (85)	3.4 (44)	3.5 (42)	
Used Street Drugs										
Used Street Drugs &	5.5 (100)	2.4 (44)	3.2 (61)	2.1 (52)	1.9 (40)	3.0 (36)	0.6 (9)	1.0 (13)	1.0 (12)	
Missing Street Drug Use Data	2.9 (52)	2.0 (38)	3.0 (56)	3.8 (92)	3.0 (64)	5.7 (201)	2.1 (30)	2.2 (29)	3.6 (43)	
Pre-existing Diseases										
Chronic Hypertension	1.1 (20)	1.2 (23)	0.9 (17)	1.2 (30)	0.7 (16)	0.7 (24)	1.2 (17)	0.8 (10)	1.1 (13)	
Renal Disease	0.3 (5)	0.2 (3)	0.0 (0)	0.1 (3)	0.0 (0)	0.1 (2)	0.1 (1)	0.1 (1)	0.1 (1)	
Heart Disease	0.5 (9)	0.2 (4)	0.5 (10)	0.7 (16)	0.5 (11)	0.4 (15)	0.9 (13)	0.4 (5)	0.7 (8)	
Diabetes Mellitus	1.1 (20)	1.4 (26)	1.6 (30)	1.4 (35)	0.7 (16)	1.0 (34)	0.5 (7)	1.5 (19)	0.4 (5)	
Other Medical Diseases	4.0 (72)	3.6 (68)	3.7 (70)	3.3 (79)	4.4 (95)	3.3 (116)	4.3 (61)	4.1 (54)	3.9 (47)	
Problems in Current Pregnancy										
Acute Medical Problems	0.4 (7)	0.6 (12)	0.3 (6)	0.6 (14)	0.1 (2)	0.3 (10)	0.4 (6)	0.5 (7)	0.2 (2)	
Polyhydramnios	2.5 (46)	2.5 (46)	2.1 (39)	2.3 (56)	2.2 (48)	2.5 (88)	1.6 (23)	1.9 (25)	2.2 (26)	
Oligohydramnios										
Gestational Diabetes	3.3 (60)	4.2 (78)	4.2 (79)	3.3 (79)	3.5 (75)	3.9 (138)	2.8 (40)	4.1 (54)	3.3 (40)	
Nutritional Problems										
Anemia	0.6 (11)	0.5 (10)	0.3 (5)	0.3 (7)	0.5 (10)	0.5 (16)	0.6 (9)	0.5 (6)	0.0 (0)	
Poor Gestational Weight Gain	0.7 (13)	1.0 (19)	0.8 (16)	0.8 (20)	1.2 (26)	1.8 (64)	1.3 (19)	0.9 (12)	1.0 (12)	
Pregnancy Complications										
Gestational Hypertension	3.3 (59)	4.2 (79)	3.3 (62)	4.0 (98)	3.4 (73)	4.0 (139)	4.6 (65)	4.2 (55)	4.8 (57)	
Gestational Hypertension and Proteinuria	0.8 (15)	1.2 (22)	1.0 (19)	0.9 (21)	1.1 (24)	1.3 (44)	0.5 (7)	0.9 (12)	1.5 (18)	
Bleeding < 20 Weeks	3.8 (69)	4.4 (82)	2.8 (53)	4.4 (83)	5.3 (113)	2.8 (97)	3.5 (50)	3.7 (48)	6.2 (74)	
Bleeding ≥20 Weeks	3.3 (59)	2.8 (53)	2.9 (55)	2.7 (65)	3.9 (84)	2.5 (86)	2.1 (30)	3.1 (41)	3.0 (36)	
Placenta Previa	0.1 (2)	0.2 (4)	0.3 (6)	0.2 (6)	0.5 (10)	0.5 (16)	0.3 (4)	0.2 (3)	0.3 (3)	
Ruptured Membranes	3.4 (61)	3.0 (56)	3.2 (61)	3.0 (72)	3.4 (74)	3.0 (105)	2.5 (35)	3.1 (4)	2.9 (35)	

Public Health Service Area										
astle Downs % (N) (1306)	West (South) % (N) (1195)	South West (W) % (N) (1407)	South West (E) % (N) (1485)	South Central % (N) (1649)	Leduc % (N) (470)	Beaumont % (N) (260)	Sherwood Park % (N) (1299)	Strathcona County % (N) (531)	Thorsby % (N) (229)	PHSA 18 % (N) (1387)
5.1 (67)	5.4 (65)	1.4 (20)	4.4 (66)	5.6 (93)	8.5 (40)	2.3 (6)	2.2 (28)	3.0 (16)	7.9 (18)	14.6 (203)
83.2 (1086)	78.2 (934)	73.9 (1039)	78.8 (1170)	78.2 (1289)	83.8 (394)	82.3 (214)	83.7 (1088)	78.9 (419)	81.2 (186)	73.6 (1020)
11.7 (153)	16.4 (196)	24.7 (348)	16.8 (249)	16.2 (267)	7.7 (36)	15.4 (40)	14.1 (183)	18.1 (96)	10.9 (25)	11.8 (164)
3.2 (42)	1.5 (18)	1.7 (24)	2.4 (35)	2.1 (34)	1.1 (5)	0.8 (2)	1.0 (13)	1.3 (7)	0.9 (2)	2.2 (30)
0.2 (2)	0.3 (4)	0.5 (7)	0.9 (14)	0.3 (5)	0.4 (2)	0.8 (2)	0.4 (5)	0.2 (1)	0.4 (1)	0.4 (6)
6.9 (90)	5.4 (65)	4.4 (62)	6.3 (94)	8.3 (137)	8.3 (39)	8.8 (23)	9.2 (119)	9.4 (50)	12.2 (28)	7.9 (109)
28.8 (376)	31.1 (372)	27.4 (385)	28.6 (425)	30.0 (494)	28.1 (132)	25.8 (67)	26.9 (350)	29.2 (155)	24.0 (55)	32.2 (447)
4.1 (53)	3.1 (37)	3.7 (52)	3.1 (46)	3.0 (49)	3.8 (18)	5.0 (13)	4.2 (55)	4.1 (22)	2.6 (6)	5.2 (72)
1.4 (18)	1.5 (18)	0.7 (10)	1.3 (19)	1.3 (21)	0.6 (3)	1.9 (5)	1.2 (16)	1.1 (6)	2.2 (5)	1.8 (25)
0.6 (8)	0.9 (11)	0.3 (4)	0.7 (10)	0.4 (7)	0.6 (3)	0.8 (2)	0.5 (6)	1.5 (8)	0.4 (1)	0.8 (11)
0.9 (12)	0.4 (5)	0.6 (9)	0.4 (6)	0.5 (8)	0.6 (3)	1.5 (4)	0.4 (5)	0.2 (1)	0.0 (0)	0.6 (8)
41.6 (544)	44.5 (532)	47.7 (671)	47.1 (700)	49.5 (816)	41.5 (195)	28.5 (74)	38.7 (503)	37.7 (200)	34.1 (78)	45.0 (625)
56.0 (731)	53.7 (642)	51.3 (722)	50.8 (754)	49.5 (816)	56.8 (267)	68.0 (177)	59.9 (778)	59.7 (317)	64.6 (148)	51.3 (711)
2.4 (31)	1.8 (21)	1.0 (14)	2.1 (31)	1.0 (17)	1.7 (8)	3.5 (9)	1.4 (18)	2.6 (14)	1.3 (3)	3.7 (51)
62.7 (819)	51.6 (617)	40.8 (574)	46.9 (696)	45.9 (757)	48.5 (228)	48.1 (125)	43.7 (568)	48.8 (259)	64.2 (147)	65.5 (908)
7.0 (92)	11.0 (131)	19.2 (270)	19.2 (285)	17.7 (292)	21.1 (99)	25.8 (67)	19.6 (254)	23.4 (124)	15.3 (35)	12.6 (175)
11.9 (155)	7.4 (89)	6.8 (96)	6.2 (92)	8.1 (134)	9.6 (45)	6.2 (16)	6.2 (81)	8.5 (45)	7.9 (18)	16.4 (228)
17.6 (230)	7.5 (90)	6.4 (90)	5.5 (82)	7.0 (115)	10.2 (48)	5.4 (14)	6.5 (85)	4.3 (23)	14.4 (33)	14.5 (201)
8.1 (106)	8.8 (105)	6.5 (91)	7.7 (114)	7.7 (127)	6.0 (28)	6.5 (17)	6.5 (85)	8.9 (47)	9.6 (22)	9.6 (133)
23.4 (306)	20.5 (245)	8.7 (122)	17.8 (264)	23.6 (389)	30.9 (145)	19.2 (50)	17.3 (225)	20.2 (107)	28.8 (66)	41.7 (578)
3.4 (44)	3.5 (42)	3.3 (47)	3.2 (48)	4.8 (79)	6.0 (28)	2.3 (6)	2.8 (37)	4.0 (21)	3.1 (7)	9.4 (130)
1.0 (13)	1.0 (12)	0.6 (9)	0.9 (13)	1.5 (25)	1.3 (6)	0.4 (1)	0.7 (9)	0.8 (4)	0.9 (2)	7.5 (104)
2.2 (29)	3.6 (43)	4.1 (57)	4.2 (62)	4.3 (71)	7.0 (33)	5.4 (14)	3.9 (51)	7.2 (38)	5.7 (13)	4.4 (61)
0.8 (10)	1.1 (13)	0.9 (13)	0.9 (14)	0.2 (4)	0.9 (4)	0.4 (1)	0.8 (10)	0.4 (2)	0.9 (2)	1.2 (17)
0.1 (1)	0.1 (1)	0.1 (2)	0.1 (1)	0.1 (2)	0.0 (0)	0.0 (0)	0.1 (1)	0.2 (1)	0.0 (0)	0.3 (4)
0.4 (5)	0.7 (8)	0.6 (9)	0.3 (4)	0.5 (9)	0.4 (2)	1.2 (3)	0.5 (7)	0.6 (3)	1.3 (3)	1.0 (14)
1.5 (19)	0.4 (5)	0.8 (11)	0.9 (14)	0.7 (12)	0.9 (4)	0.4 (1)	0.4 (5)	0.9 (5)	0.4 (1)	0.4 (6)
4.1 (54)	3.9 (47)	3.8 (54)	3.5 (52)	3.0 (50)	3.2 (15)	4.6 (12)	3.1 (40)	2.3 (12)	4.4 (10)	4.0 (56)
0.5 (7)	0.2 (2)	0.5 (7)	0.5 (7)	0.2 (4)	0.6 (3)	0.4 (1)	0.2 (3)	0.2 (1)	1.3 (3)	0.4 (6)
1.9 (25)	2.2 (26)	2.2 (31)	1.9 (28)	2.4 (39)	1.1 (5)	2.7 (7)	2.7 (35)	1.9 (10)	1.7 (4)	3.0 (42)
4.1 (54)	3.3 (40)	2.9 (41)	3.5 (52)	3.9 (64)	1.3 (6)	3.8 (10)	1.9 (25)	3.2 (17)	3.5 (8)	2.6 (36)
0.5 (6)	0.0 (0)	0.0 (0)	0.1 (2)	0.0 (0)	0.4 (2)	0.0 (0)	0.1 (1)	0.2 (1)	0.0 (0)	0.8 (11)
0.9 (12)	1.0 (12)	0.5 (7)	1.5 (22)	1.1 (18)	0.9 (4)	0.8 (2)	0.8 (11)	0.9 (5)	2.2 (5)	1.6 (22)
4.2 (55)	4.8 (57)	3.6 (50)	3.8 (57)	3.3 (55)	3.2 (15)	3.1 (8)	4.5 (59)	4.9 (26)	3.1 (7)	4.2 (58)
0.9 (12)	1.5 (18)	1.1 (16)	1.3 (19)	1.2 (20)	1.3 (6)	0.4 (1)	1.3 (17)	0.9 (5)	0.9 (2)	1.4 (20)
3.7 (48)	6.2 (74)	3.8 (53)	4.6 (69)	3.0 (49)	3.8 (18)	3.8 (10)	3.3 (43)	2.8 (15)	4.8 (11)	3.5 (49)
3.1 (41)	3.0 (36)	2.6 (37)	2.6 (39)	2.5 (41)	2.1 (10)	1.5 (4)	2.2 (28)	1.7 (9)	3.1 (7)	3.5 (48)
0.2 (3)	0.3 (3)	0.1 (2)	0.3 (4)	0.4 (7)	0.4 (2)	0.4 (1)	0.2 (2)	0.4 (2)	0.4 (1)	0.1 (2)
3.1 (4)	2.9 (35)	2.9 (41)	3.6 (54)	2.5 (42)	2.3 (11)	2.3 (6)	3.2 (41)	1.1 (6)	3.1 (7)	4.6 (64)